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SURGICAL TREATMENT OF SEVERE ORCHITIS IN MUMPS*

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THE early surgical treatment of severe orchitis of mumps accomplishes prompt relief of pain and saves the testicle from subsequent atrophy. The operation must be done before the testicular tissue has undergone pressure necrosis, but should be reserved for cases in which the severity of the symptoms warrants such interference.

In 1864, Henry Smith[1] advised multiple punctures of the tunica albuginea for orchitis; he stated that this operation had been in vogue in Paris for at least two years. One finds references to this work in the literature on mumps, but the orchitis so treated was of gonorrheal origin. Comby,[2] in his book on mumps published in 1893, makes no mention of any surgical treatment of orchitis. Schottmüller,[3] in 1904, advised incision through the tunica vaginalis for release of the hydrocele fluid in mumps orchitis. In 1912, G. G. Smith[4] treated 2 patients with mumps orchitis by incising the tunica albuginea, and in 1920, Ballenger and Elder[5] reported on 3 patients so treated.

Edema is characteristic of the pathology of an invasion of gland tissue by the mumps virus. The dense fibrous layer covering the testicle offers resistance to this swelling. If the edema develops rapidly, the internal pressure causes intense pain, often associated with backache. Repeated chills, with high fever, nausea, vomiting and even delirium, may take place. The pain may be so excruciating that ½ gr. of morphine fails to give relief, and so tender is the part that the patient objects to the most gentle examination. This situation may develop rapidly in fulminating cases, in which the testicle swells rapidly and hydrocele fluid forms. The scrotum becomes markedly edematous and bluish red. In other cases, the testicle may gradually reach double its normal size in forty-eight hours, with only moderate discomfort, slight tenderness, no edema of the scrotum and only a moderate fever. It may subside gradually, when no surgical interference is necessary, or it may suddenly develop into the fulminating picture described above.

Although mumps is manifested by a parotitis in the vast majority of cases, it is well to keep in mind that the parotids may show no swelling, and that the submaxillary and sublingual glands may take the brunt of the attack. Furthermore, the involvement of the salivary glands may follow other manifestations of the disease. Thus, an orchitis, a pancreatitis or an encephalitis may precede, accompany or follow the parotitis. Finally, the only manifestation of mumps may be in one of these organs remote from the salivary glands. Here, the diagnosis is based on the circumstantial evidence of exposure, the incubation period of approximately eighteen days and the course pursued. In 1920, Wesselhoeft[6] collected from the literature 30 cases of primary orchitis and 64 cases in which orchitis occurred without involvement of the salivary glands. Since then, several more have been reported. During epidemics, such cases crop out, and to add to the difficulty, they may occur in patients who have had mumps previously. However, one attack of the disease, even though it affects only one salivary gland, usually conveys a lasting immunity.[7, 8]

Orchitis very rarely occurs before the age of puberty, but such cases have been recorded. At and above that age, its incidence in mumps is 18 per cent. Collected reports from the literature indicate that under epidemic conditions both testicles are likely to be involved in 1 out of 6 cases.[6]

In the follow-up examinations of 347 cases, atrophy was revealed in 190, or 54.7 per cent.[6] Sterility as a result of atrophy is actually rare.[7] In

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the unilateral cases, the unaffected testicle serves adequately for procreation. In the bilateral cases, enough undamaged tubules remain to supply spermatozoa, but probably not in the usual abundance, and thus reduce the chances of conception to an indeterminable degree. Sterility is not to be confused with impotence. As pointed out by Stengel,[9] sterility implies the lack of sufficient sex cells, whereas impotence refers to the inability to complete the sexual act. Fear of sterility after mumps orchitis sometimes leads to a mental complex that may promote impotence.

A description by Wolbach of the pathology of mumps orchitis is incorporated in the paper by G. G. Smith.[4] In the biopsy specimen, it was found that the process did not affect the testicular tissue uniformly. Some groups of seminiferous tubules were completely destroyed. The interlobular connective tissue throughout was edematous, and between the tubules there were fibrin, small areas of hemorrhage, and many polymorphonuclear and endothelial leukocytes. Manca[10] subsequently confirmed these findings in an autopsy specimen. The pathology of the parotitis in the experimental disease in monkeys has been described by Johnson and Goodpasture,[11] who found edematous swelling of the gland with pin-point hemorrhages and destructive changes through the parenchyma, including disintegration of the acinar cells and the presence of cytoplasmic inclusions. There was an infiltration of mononuclear phagocytes, and later of lymphocytes, into the necrotic areas and about the ducts. The authors found that healing took place through removal of the debris and regeneration of acinal epithelium, which restored the gland without scarring. In these two descriptions, there is a contrast that is of importance to the prognosis. Wolbach described a well-advanced orchitis in which the inflammation had called forth polymorphonuclear cells. In severe orchitis, the blood counts tend to shift from a lymphocytosis to a polymorphonuclear leukocytosis. This shift in the blood picture is comparatively rare in parotitis, involvement of the submaxillary glands, pancreatitis, encephalitis and oöphoritis. The reason for this rise in neutrophils seems to be a true pressure necrosis in the testicle, in addition to the benign and reparable inflammatory process seen in other organs. Furthermore, in no other gland does mumps invasion result in atrophy, with the possible exception of the prostate.[12]

The pathology in a totally atrophied testicle has been described by Stolz.[13] The resistance of the tunica albuginea appears to be responsible for the severe pain and the pressure necrosis that leads to subsequent testicular atrophy.

The virus of mumps may invade the seminiferous tubules of the testicle, the epididymis and the vas deferens in varying degrees of severity, either simultaneously or separately. Moreover, in rare cases, the prostate and the seminal vesicles may also be involved.

Schottmüller[3] recommended incision through the wall of the scrotum for the purpose of releasing the hydrocele fluid. This operation, however, did not relieve the pressure necrosis going on within the testicle, and therefore accomplished little or nothing. In our experience, hydrocele fluid increases more rapidly from an epididymitis than from an orchitis. However, if either of these conditions exists in a severe form, the other organ also becomes involved to a certain degree. The epididymitis, which is relatively less painful than that caused by gonococcal infection, is as self-limited as the swelling of the parotid. As the inflammation in the epididymis diminishes, the hydrocele recedes spontaneously. The only possible value, then, of an incision through the scrotum is to expose the testicle and thus to determine the situation more accurately, especially when the diagnosis is in doubt. In primary cases, the question of differentiation from an early torsion of the testicle may arise; therefore, a view of the testicle may be desirable.

At the onset of orchitis, the scrotal wall is thin, so that one can easily palpate the testicle and any enlargement of the epididymis. If the testicle does not yield to pressure, presents a stonelike hardness and is very tender, it is wise to operate without further delay. As the condition progresses into the severe stage, the actual size and firmness of the testicle become more obscured by the edema of the scrotum and the accumulation of hydrocele fluid.

An incision through the scrotum, which in these cases may be 1 cm. thick from edema, causes a spurt of yellow hydrocele fluid. This spurt may rise like a fountain toward the face of the operator, particularly if a small nick is made in the tunica vaginalis. The incision is enlarged with scissors to about 3 cm., thus exposing the surface of the testicle to view. There is no necessity of delivering the testicle as done by Smith[4] and Ballenger and Elder.[5] It may be turned within the scrotum to demonstrate the condition of the epididymis. In severe cases, the tunica presents a surface that does not yield to pressure and may be studded with small petechiae. The tunica albuginea is incised longitudinally to a length of 1.5 cm. The internal pressure may cause the testicular tissue to protrude through the incision. The greater the tension, the less the bleeding. Care is taken to avoid any outwardly visible blood vessel, and

small spurters may be secured by fine ligatures. Another incision, crosswise to the first but beside it, may be made to relieve tension in both directions. The scrotal incision is then stitched with silk, a small rubber-tissue drain or simply an open end of the wound being left to afford an exit for the profuse drainage that follows. The dressing should be secured by a T bandage, with two lines coming up over the groins. This dressing should be changed within two hours, and at frequent intervals through the first twenty-four or forty-eight hours, or as indicated by the amount of drainage.

Nitrous oxide and oxygen is the anesthetic of choice. We attempted to use local anesthesia in one case but without success, because the operation was much too painful. This was the only patient who expressed dissatisfaction with the operation at the follow-up examination. In another case, we used intravenous Pentothal Sodium, but this patient had a temperature of 103°F. at the time and squirmed about during the operation, although entirely unconscious. Furthermore, he remained in a deep sleep for two hours. With nitrous oxide and oxygen, the patient comes out of the anesthetic immediately after the operation. It is surprising how promptly the tenderness of the testicle subsides. While the first dressing is being changed, the incised testicle may be gently palpated without causing undue pain, whereas prior to the operation even the gentlest possible handling is barely tolerated.

There is an immediate feeling of relief after the operation. The temperature drops sharply. One of our patients with a temperature of 105°F. and a mild delirium at the time of the operation was mentally clear half an hour later. The temperature fell to 100°F. in four hours, and an uneventful recovery followed.

The stitches in the scrotum should be left in for one week, since there is a tendency for the wound to gape if they are removed earlier. When the operation is done within forty-eight hours of the onset, the swollen testicle may go down to almost normal size within a week, but more frequently this takes ten days to two weeks. One patient who was operated on late but had high fever still had an enlarged testicle after a month. On the follow-up examinations six months and one year later, this case showed some atrophy to have taken place, the testicle being noticeably smaller than the unaffected one; the scar was barely visible in the folds of the scrotum. G. G. Smith[4] did not report on the follow-up of his 2 cases. Ballenger and Elder[5] incised the tunica albuginea in 3 cases, and on the follow-up examinations twenty months to three years later, there was no evidence of atrophy.

Twelve patients with severe mumps orchitis were operated on at the Haynes Memorial Hospital (Table 1). Nine of these were followed up six months and one year later. In only 1 of the 9, the patient described above as being operated on late, was there any observable atrophy of the testicle when compared with the unaffected one. In a case followed at a boarding-school infirmary, the second testicle became involved after the operation on the first; no atrophy took place. Thus, ten testicles were operated on early, with no subsequent atrophy, and one was operated on late, with subsequent partial atrophy.

TABLE 1. *Summary of Results of Treatment of Mumps Orchitis.*

TYPE OF CASE	TOTAL NO. OF CASES	TESTICLES INVOLVED	NO. OF CASES FOLLOWED	TESTICLES WITH ATROPHY	TESTICLES WITHOUT ATROPHY
Severe (operation)	13	14	10	1*	10
Mild (no operation)	14	17	10	0	13
Totals	27	31	20	1	23

*Late operation.

With the exception of 1 patient operated on in 1929, all occurred in the years 1936–1941 inclusive. During this period, there were 10 cases of mild mumps orchitis at the Haynes Memorial Hospital. Four other mild cases were followed outside the hospital. These patients were not operated on. Follow-up examinations in 10 of these 14 cases revealed no atrophy. The absence of any evidence of atrophy after these mild cases is not in accord with an assertion by Stolz,[13] based on reports of other observers, that the intensity of the inflammation during the orchitis is not a reliable indication of the amount of atrophy likely to ensue.

We have stressed the rapid fall in the temperature following operation. This statement needs qualification because the involvement of another organ may retard the fall in the temperature or subsequently cause a sharp rise. In 1 case, the second testicle became severely involved and was operated on two days after the first operation. Both testicles were normal in size at the follow-up examination. In 2 severe cases that came to operation, there was an accompanying encephalitis. In 2 of the mild cases

without operation, an encephalitis was the predominant complication. In 1 of these, the temperature rose abruptly to 107°F., accompanied by delirium, when the orchitis was superimposed on the encephalitis, but the testicle was never seriously threatened, and the temperature fell rapidly throughout the night. Aside from a lumbar puncture, there was no surgical interference, and no atrophy of the testicle followed. In 1 case, there was well-marked tenderness over the region of the pancreas, and the temperature was elevated.

The value of heat, cold and other applications, as well as morphine, is, in our opinion, beside the point in severe orchitis, as is any acute condition in which surgery is indicated. The use of convalescent serum is as illogical as it is ineffective. Whether the use of this serum in a case of mumps will prevent orchitis is a debatable question,[14] and outside the scope of this paper. However, there is no doubt that convalescent serum is a valuable preventive against mumps when used soon after exposure.[14]

Summary

The rationale of incising the tunica albuginea in severe orchitis is somewhat comparable to that of paracentesis of a bulging, painful drumhead in otitis media. The operation must be done sufficiently early to avoid pressure necrosis in the testicle. It is useless to operate when the process is already on the wane. Early operation relieves pain, reduces the fever and avoids atrophy. It is not indicated in mild cases or those in which an epididymitis predominates. An enlarged, hard, tender testicle, with chills and fever, constitutes the indication for surgical interference. Nitrous oxide and oxygen is the anesthetic of choice. A rise in temperature after operation signifies an invasion of the virus in some other organ.

The end results of 9 hospital cases and 1 operated on elsewhere are presented. Moderate atrophy took place in the case operated on too late. In the others, there was immediate relief, and no atrophy was apparent at follow-up examinations. Fourteen cases of mild orchitis were observed during this period. The patients were not operated on. A follow-up examination on 10 of these revealed no atrophy. The expected incidence of atrophy after mumps orchitis without surgical interference is 54.7 per cent. Our experience suggests that atrophy occurs only after severe cases, and that by an early operation in such cases atrophy can be avoided.

References

1. Smith, H. On the treatment of acute orchitis by puncturing the testicle. *Lancet* 2:149, 1864.
2. Comby, J. *Les Oreillons*. 203 pp. Paris: Ruess et Cie., 1893.
3. Schottmüller, H. Parotitis Epidemica. In *H. Nothnagel's Specielle Pathologie und Therapie*. Vol. 3, Pt. 2, Sect. 3. Vienna: Alfred Hölder, 1904. P. 49.
4. Smith, G. G. Two cases of orchitis due to mumps treated by operation. *Boston M. & S. J.* 167:323-325, 1912.
5. Ballenger, E. G., and Elder, O. F. Orchitis from mumps: the need of conserving the testes by incision of the tunica albuginea. *J. A. M. A.* 75:1257, 1920.
6. Wesselhoeft, C. Orchitis in mumps. *Boston M. & S. J.* 183:458-461, 491-494, 520-524, 1920.
7. *Idem*. Mumps. In Christian's *The Oxford Medicine*. 5:489-497 (36). New York: Oxford University Press, 1941.
8. Gordon, J. E., and Heeren, R. H. The epidemiology of mumps. *Am. J. M. Sc.* 200:412-428, 1940.
9. Stengel, A., Jr. Mumps orchitis. *Am. J. M. Sc.* 191:340-356, 1936.
10. Manca, C. Über die Mumpsorchitis. *Virchow's Arch. f. path. Anat.* 285:426-442, 1932.
11. Johnson, C. D., and Goodpasture, E. W. The histopathology of experimental mumps in the monkey, *Macacus rhesus*. *Am. J. Path.* 12:495-510, 1936.
12. Robinson, W. J. Prostatic atrophy and mumps. *M. Rec.* 187:404, 1915.
13. Stolz, A. Über Totalnekrose des Hodens bei Mumps. *Mitt. a. d. Grenzgeb. d. Med. u. Chir.* 7:392-406, 1901.
14. Wesselhoeft, C., and Walcott, C. F. Mumps as a military disease and its control. *War Medicine* 2:213-222, 1942.

ECLAMPSIA

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THE purpose of this paper is to review some aspects of eclampsia and to present additional statistics.

ETIOLOGY

The cause of eclampsia remains unknown, in spite of many theories.

Placental infarcts have frequently been discussed as a cause of eclampsia. Recently, Hunt and his associates[1] suggested that placental infarcts and degenerating placental tissue liberate autolysates, which, if massive or if renal function is poor, produce severe late toxemia or eclampsia. Patterson et al.[2] caused acute placental infarctions, convulsions and death at term in rabbits by total thyroidectomy at the end of the first trimester of pregnancy. They believe that hypercholesteremia of the fetus resulted in arterial disease, which accompanied fetal hypometabolism, and that placental infarctions and convulsions were due to split-protein products from the infarcted placenta.

Bartholomew and Kracke[3] studied a thousand placentas and found acute infarcts in all those from patients who had pre-eclampsia or eclampsia. In their opinion, the infarcts are due to cholesterol-induced arterial disease and the eclampsia to the absorption of split-protein products from the infarcted area. They produced convulsions and death in guinea pigs by the injection of placental decomposition products.

Peters et al.[4] found that 41, or 13 per cent, in a series of 320 patients with toxemia had had pyelitis or pyelonephritis, which, according to these authors, led to toxemia by producing renal damage and diminishing renal function. However, Mussey and Lovelady,[5] in a series of 163 patients with pre-eclamptic toxemia or eclampsia, found 6 or 7 who showed symptoms of pyelitis in the puerperium. None gave a history of pyelitis before the first pregnancy, and among 117 cases of pyelitis of pregnancy, 3 patients developed acute hypertensive eclampsia and 1 died of an inflammatory kidney lesion. Prather and Sewall,[6] studying 72 patients with pyelonephritis during pregnancy who returned to the hospital with a subsequent pregnancy, found that 13 per cent had toxemia. They point out that 10 per cent of all pregnant women have toxemia.

Strauss[7] observes that 80 per cent of the women said to have toxemia actually have chronic vascular or renal disease before and after the gravid state, and that another 5 per cent have such disease in acute form. The remaining 15 per cent have toxemia and, under proper care, will have normal succeeding pregnancies; he calls this group the "water-retention toxemias." Such toxemia may be caused primarily by low plasma proteins, excessive sodium intake or, in many cases, both. Anemia, primary renal failure and a rise in intercapillary pressure, as with congestive heart failure or venous obstruction, are conducive to water retention.

Smith and Smith[8] show that the urinary excretion of estrogenic compounds from the fifth month of pregnancy to term in pre-eclampsia and eclampsia is considerably less than it is in normal pregnancy. In their cases, the level of serum estrogenic substance also failed to show the rise characteristic of normal pregnant women. Venning[9] and Weil[10] state that the excretion of pregnanediol in normal pregnancy gradually increases until term, but in toxemia there is a sharp decrease. Smith, Smith and Pincus[11] show that the ratio of estriol to estrone is about 15:1 in the last month of normal pregnancy, but in pre-eclampsia and eclampsia, this ratio is considerably lower. Pincus and Zahl[12] indicate that the corpus-luteum hormone, progesterone, facilitates the conversion of estrone into estriol, and that the excretion of sodium pregnanediol glucuronide reflects the action of progesterone. The urinary chorionic gonadotropic hormone in normal pregnancy has a peak of excretion in the early months (the twentieth to the fiftieth day after the missed menstrual period, according to Evans et al.,[13] and at about two months according to Smith and Smith[8]); after this peak is reached, the excretion decreases rapidly and remains constant until term.

Smith and Smith [14, 15] have shown that the placentas from pre-eclamptic and eclamptic patients contain an abnormally high concentration of prolan, and that the estrogenic content of these placentas is low; furthermore, the serum prolan is usually high in pre-eclampsia and eclampsia. They found that low or decreasing pregnanediol and estriol, a high or increasing percentage of estrogenic activity accountable to estradiol, decreasing or absent estrone, and high or increasing ratios of estrogenic potency after hydrogenation characterized the urines of every toxemic case. They believe that toxemia of pregnancy is closely associated with a progressive deficiency of progestin, a concomitant decrease of estrogen and a

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consequent changed metabolism of estrogen involving greater and more rapid destruction.

Weiss et al.,[16] in a study of a series of autopsies on women who died of eclampsia, noted rapid improvement after the uterus was emptied. In 16 cases in the series, death of the fetus in utero did not result in improvement of the patient. After fetal death, hormones originating in the placenta continued to be eliminated in the urine. The authors believe, since there is no valid evidence that pressure on the ureter or renal artery, primary kidney disease, infection or nutritional disease is primarily responsible for the syndrome, that the presence of a functioning placenta maintains the syndrome and that removal of the placenta is responsible for the improvement. No identifiable chemical substance was found to be responsible. Extracts of placentas from normal or toxemic pregnant women failed to yield pressor substances. Weiss and his associates do not believe that the syndrome occurs spontaneously or has been induced in animals. Their experiments in nonpregnant and pregnant rabbits and rats with Pitressin, estrogenic substances, progesterone, pregnancy-urine prolan, desoxycorticosterone and placental extracts failed to produce the syndrome. They consider the changes observed in the kidneys and other organs during the course of the syndrome secondary to the suspected chemical or "hormonal" changes, but in severe cases this secondary damage to the kidneys and liver can play a role in the vicious circle leading ultimately to convulsions, acidosis and circulatory collapse. They believe that the primary factor initiating the syndrome originates from the placenta; it may be a normal hormonal constituent of pregnancy in abnormal amounts, or in normal amounts acting on a vascular system with a congenital or acquired hyperirritability.

It seems, from the theories discussed, that whatever its cause, eclampsia affects first, or even arises from, the placenta. The chief pathologic damage is to the vascular system. And, possibly, it may have a hormonal origin.

Physiologic Pathology

A major abnormality during pregnancy is sudden increase in weight due to edema. Water retention can be caused by changes in sodium balance brought about by a high salt intake, or by a large amount of sodium bicarbonate taken for "heart burn."[7] Anemia, for an unknown reason, is also conducive to water retention. Hormone balance plays a part in water retention, and vitamin lack, diabetes and anything else that disturbs the blood supply to the placenta may cause hormonal imbalance. Decrease in plasma protein is another important factor in water retention; the albumin fraction is four times as active osmotically as the globulin fraction, so that it is essential to do separate analyses. Hydremia, rise in intracapillary pressure, hypothyroidism, primary renal failure and cardiac failure are other factors. Weiss et al.[16] find that 64 per cent of all normal pregnant women have generalized edema of varying degree. They believe that the above factors are not the primary causes of edema, although they may play a secondary role. They suspect that, by exclusion, a chemical or hormonal etiology, resulting in retention of water and electrolytes in the tissues, is the cause. Dieckmann[17] states that about 75 per cent of eclamptic women have edema. In his cases, edema was not due to hypoproteinemia. The osmotic pressure was above edema level, and the serum albumin and globulin were normal in composition. He believes the edema is due to changes in permeability of the capillary and cell walls.

Hypertension, another serious abnormality, can be caused by vascular spasm as well as by water retention. Hypertension occurring before pregnancy is usually due to one of the nephritides.

Headache and visual disturbances are believed to be the results of cerebral edema, which follows edema as considered above, and of ischemia of the brain.

Pathology

Irving[18] gives a thorough review and presents additional descriptions of the pathological findings in eclampsia; he renews the belief that arteriolar derangement affecting all the terminal arterioles is the primary factor in the disease. The organs most affected are the kidneys. He gives Fahr's description of an increase in the size of the glomeruli due to swelling of the capillary walls, and a relative absence of blood cells in the capillary lumens, producing marked local ischemia. Hyaline thrombi are found within the loops. The tubules exhibit albuminous degeneration, which may advance to hyaline formation and fatty changes. Irving states that all glomeruli are not always equally involved; in some cases, individual ones may escape entirely, and the tubules draining these glomeruli are also unaffected. He believes that spasm of the afferent arterioles is the cause of this damage. Bell[19] first demonstrated a massive thickening of the capillary basement membrane. Weiss et al.,[16] describing kidney changes, discovered that the lesions were primarily degenerative and believed that the term "glomerulonephrosis," which has been applied to this condition, seems to be most suitable. They found hyalinization of the

arterioles in some cases. There were no specific changes in the interlobular and arciform arteries.

The liver lesions of hemorrhage, necrosis and fatty degeneration — usually predominant in the periphery of the lobule but also found in the central and midzonal portions — are reviewed by Irving,[18] who places the liver damage on a vascular basis, since thromboses of the radicles of the portal vein or of the small branches of the hepatic artery in the portal space may be observed. Weiss et al.,[16] in autopsies on 25 women who died of eclampsia, noted liver lesions in less than half the cases. The lesions varied from mild to severe necrosis and hemorrhage in the peripheral lobules.

Irving[18] describes hemorrhages in the cortex of the adrenal glands and in the capsule and parenchyma of the spleen. In some cases, necrosis in the lymphoid follicles was noted in the spleen; in others, acute inflammation in the spleen was found. Thickening of the arterioles has been observed.

Evidence of arteriolar spasm in the general peripheral circulation, as seen in the capillary loops at the bases of the nails and in the retinal vessels, is also discussed by Irving, and he states that the brain shows ischemic cellular disease, as well as vascular spasm causing necrosis and rupture of the vessel walls.

According to Hertig,[20] 50 per cent of uteri show bilateral cornual and subserosal hemorrhages due to arteriolar spasm. He describes the rare "eclampsia of the uterus," that is, toxic separation. He has found capillary thrombosis, petechial hemorrhage and edema at one time or another in all the organs; 60 per cent of eclamptic placentas are infarcted.

Tenney and Parker[21] describe placental lesions consisting of a premature aging of the placenta. Although 10 to 50 per cent of a normal placenta shows syncytial degeneration, the amount of degeneration in eclampsia is over 50 per cent. This degeneration consists of a clumping together and autolysis of nuclei in the syncytial cytoplasm, clumps of darkly staining masses with no cell outline, and areas of syncytial cells without nuclei. The final stage is the disappearance of all nuclei from the syncytial layer; the villus is surrounded by a thin, irregular mass of hyaline material. Moreover, there is marked congestion of the blood vessels of the villi in the toxemic cases. The blood pressure and the albuminuria closely correlated with the placental findings in 90 per cent of the cases.

Statistics

At the Massachusetts Memorial Hospitals, from January 1, 1923, to November 1, 1941, there were 55 cases of eclampsia — emergency, clinic and private — among 22,089 hospital and district deliveries, an incidence of 0.249 per cent, or 1:402. There were 12 maternal deaths, a mortality of 22 per cent (Table 1). Among the fatal cases, there were 2 vaginal sections, 1 classical cesarean section, 1 manual dilatation and 1 high-forceps delivery of a large baby through a generally contracted pelvis — this last patient, an emergency case, entered the hospital fully dilated and had several convulsions before and after delivery. Four patients died undelivered, 1 died of general peritonitis after a spontaneous delivery following artificial rupture of membranes to induce labor, and 2 died after normal deliveries following conservative treatment. From 1923 through 1931, when operative deliveries were prevalent, there were 32 cases of eclampsia and 9 deaths, a mortality of 28 per cent. Since then, the treatment has been conservative, and there have been 23 cases of eclampsia and 3 deaths, a mortality of 13 per cent. Twenty-nine of the patients were under thirty years of age, and 8 of these died, the mortality being 28 per cent; 26 were thirty years old or older, and 5 died — a mortality of 19 per cent.

Table 1. *Mortality in Cases of Eclampsia.*

Source	No. of Cases	Incidence	Maternal Mortality	Infant Mortality
		%	%	%
Massachusetts Memorial Hospitals	55	0.249	22	36 43*
Weintraub[22]	63	0.2	14	33
Ware and Noblin[23]	105	2.02	15	24
Teel and Reid[24]	173	0.312	27	51
Torpin and Coppedge[25]	350		13	31
Peckham[26]	127		11	40 51*
Chesley and Somers[27]	169	0.395	7	
Rucker[28]	204		16†	
Lazard[29]	225		13	
Davis and Harrar[30]	879	0.576	15‡	35
Schwarz and Dorsett[31]	186		7	24
Kimbrough and Shirey[32]	43	0.34	14	28
Bryant and Flemming[33]	120		2	
Greenhill[34]	78	0.28	8	28
Williams[35]	385	0.98	17	

*Excluding babies born to mothers in post-partum eclampsia.
†Last 108 cases, mortality 5.6 per cent.
‡Last 149 cases.

Prenatal care is a valuable means of reducing the incidence of and the maternal deaths from eclampsia. It is considered adequate in the clinic when a patient is seen in the fifth month of pregnancy and keeps appointments at least once every four weeks until the seventh month of pregnancy, once every two weeks until the last month, and then once weekly until delivery. It is evident from the results in various clinics that the majority of cases of eclampsia occur in the group of pregnant

women who have had no or inadequate prenatal care (Table 2).

Our statistics agree with most others in that eclampsia is predominant among primigravidas (Table 3).

There was a slight seasonal variation in the incidence of eclampsia in this series, 60 per cent of the cases occurring from October through March (Table 4). Kimbrough and Shirey[32] in a study of 43 cases, found the incidence to be as follows: "spring," 21 per cent; "summer," 21 per cent; "autumn," 35 per cent; and "winter" 23 per cent.

TABLE 2. *Prenatal Care among Patients with Eclampsia.*

SOURCE	ADEQUATE CARE	INADEQUATE CARE	NO CARE
	%	%	%
Massachusetts Memorial Hospitals	18	53	29
Kimbrough and Shirey[32]	40	27	23
Greenhill[24]	18	23	59
Ware and Noblin[23]	0	33	67

TABLE 3. *Comparison of Parity in Cases of Eclampsia.*

SOURCE	TOTAL NO. OF CASES	PRIMIGRAVIDAS	MULTIPARAS
		%	%
Massachusetts Memorial Hospitals	55	69	31
Kimbrough and Shirey[32]	43	72	28
Greenhill[24]	78	73	27
Weintraub[22]	63	57	43
Teel and Reid[24]	173	49	51
Ware and Noblin[23]	105	86	14
Total	517		
Averages		65	35

In a report of 879 cases, Davis and Harrar[30] observed that eclampsia increased through January to a high point in April, with a decrease to a low point in July.

There were two sets of twins from the 50 mothers who delivered, or 4 per cent.

There was 1 case of polyhydramnios in the 55 cases of eclampsia.

Fifty-two babies were delivered from 50 mothers, and 4 mothers died undelivered. There were 13 stillbirths, and 3 babies died after birth, making an uncorrected total of 20 deaths, a mortality rate of 36 per cent. One woman, after subsidence of convulsions and symptoms, went home undelivered, against advice.

The fetal ages when eclampsia occurred are shown in Table 5. Ware and Noblin[23] report incidences as follows: five months and under, 6 per cent; premature, 57 per cent; and full term, 37 per cent.

TABLE 5. *Fetal Ages When Eclampsia Occurred.*

SOURCE	MONTHS						
	5	6	7	7½	8	8½	9
	%	%	%	%	%	%	%
Massachusetts Memorial Hospitals	2	4	13	9	27	7	38
Weintraub[22]	0	5	16		16		63

The times that convulsions occurred are given in Table 6.

SIGNS AND SYMPTOMS

The most frequent signs and symptoms before convulsions in this series were albuminuria (96 per cent), hypertension (93 per cent), edema (82 per cent), headache (71 per cent), blurring of the eyes or spots before the eyes (38 per cent), vomiting (35 per cent) and pain (25 per cent).

The blood uric acid was recorded in 48 cases. The highest readings before, during or after convulsions in 38 patients who recovered averaged 5.8 mg. per 100 cc., and for 10 of the women who died the average was 7.1 mg. per 100 cc., in comparison with Stander's[36] figures of 5.6 and 7.6 mg. per 100 cc., respectively. Six of the recordings had readings of 3.5 mg. or below. According to Chesley,[37] the absolute level of uric acid is often elevated. More generally, the ratio of uric acid to nonprotein nitrogen is increased, the upper normal ratio being about 1:10. The cause of a rise in uric acid in eclampsia is not known. However, Cadden and Stander,[38] in a study of 5 cases, showed that eclampsia is not associated with decreased excretion of uric acid, and they believe that an elevated uric acid is probably due to impaired destruction of uric acid in the liver.

Routine determinations of the carbon dioxide combining power were not done. Stander and Radelet,[39] as well as Cadden and Stander,[38] find a decrease in the carbon dioxide combining power. Cadden and Stander emphasize the value of

TABLE 4. *Seasonal Incidence of Eclampsia.*

SOURCE	TOTAL NO. OF CASES	NO. OF CASES A MONTH											
		JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.
Massachusetts Memorial Hospitals	55	7	4 (25%)	3	3	2 (20%)	6	5	2 (20%)	4	9	2 (35%)	8
Ware and Noblin[23]	105	12	8 (28%)	9	6	14 (30%)	12	14	7 (28%)	8	4	6 (14%)	5
Teel and Reid[24]	173		(22%)			(28%)			(27%)			(23%)	

determining the carbon dioxide combining power and the uric acid. They believe that such data are more indicative of an impending development of convulsions than hypertension, amount of albuminuria or other symptoms are.

Treatment

The usual nursing routine in conservative treatment is followed. The patient is placed in a quiet, darkened room. A nurse is always in attendance and she is warned to stop the patient from choking,—because of saliva, vomitus or swallowing the tongue, — to prevent biting of the tongue, and to record the blood-pressure and pulse rates hourly. The foot of the patient's bed is elevated, if necessary. A Foley catheter is inserted, and the catheterized urine examined. Blood is taken for determinations of uric acid, carbon dioxide combining power, nonprotein nitrogen, sugar, protein and albumin-globulin ratio. Oxygen is given for cyanosis. Sodium phenobarbital, 5 gr., is given intramuscularly, and Sodium Amytal, 3 gr., is given by mouth or rectum to prevent restlessness (morphine sulfate is occasionally given). Fifty cubic centimeters of a 50 per cent glucose solution intravenously is given slowly to withdraw fluids from the edematous tissues, to increase urinary output and to combat acidosis.[40, 41] However, dehydration is not a strict rule. If there is oliguria, 500 to 1000 cc. of 10 per cent glucose in distilled water is given intravenously. This is repeated if necessary for the treatment of oliguria or concentration of the blood, according to Davis[42] and Dieckmann.[43] Davis says that when more than 200 gm. of glucose is given rapidly, it causes a very marked glycosuria. Twenty cubic centimeters of a 10 per cent magnesium sulfate solution is given intravenously at each convulsion, and between convulsions if the blood pressure starts rising. Not more than 10 gm. of magnesium sulfate has been given in twenty-four hours. Fischer[44] reports that it is safe to give up to a total of 8 gm. in twenty-four hours, as a 2.0 or 2.5 per cent solution. Torpin and Coppedge[25] give up to 24 gm. in twenty-four hours and 3500 cc. of a 5 per cent glucose solution, with good results. Fluids must be restricted in cardiac cases.

When the above treatment fails to improve the patient, a venesection is sometimes done to lower the systolic blood pressure 30 to 50 mm., 200 to 400 cc. of blood being withdrawn.

Besides the routine treatment for pulmonary edema in eclampsia, Dean[45] recommends bronchoscopy.

As soon as convulsions are controlled and the patient appears improved, and at times when the patient does not improve and it is considered desirable to avoid further delay, the membranes are ruptured. If the cervix is not sufficiently softened, it is packed for twenty-four hours. If the patient is a primipara with a long, hard cervix and a prolonged labor is feared, or if there is a cephalopelvic disproportion or any other contraindication to pelvic delivery, such as lower-segment fibroids or an amputated cervix, the patient is put in as good condition as possible, blood donors are kept available, and a cesarean section is performed.

Table 6. *Times When Eclamptic Convulsions Occurred.*

Source	Ante Partum	Intra Partum	Post Partum	Ante and Intra Partum	Ante and Post Partum	Intra and Post Partum	Ante, Intra and Post Partum
	%	%	%	%	%	%	%
Massachusetts Memorial Hospitals	42	13	18	9	5	7	5
Weintraub[32]	64	19	18				
Ware and Noblin[23]	82	2	13	1			2
Williams[35]	53	25	22				

Ware and Noblin[23] give digitalis routinely when using magnesium sulfate, because they believe that it should tend to overcome a cardiac depression produced by intravenous magnesium sulfate, since digitalis and magnesium sulfate are physiologically antagonistic in their action on the heart.

Smith and Smith[46] have used progesterone and estrogen in the treatment of toxemia, but they consider these drugs of little benefit when eclampsia has developed. They advise frequent prolan determinations and early hormonal treatment in preeclampsia. They realize that the expense of prolan determinations and hormonal treatment precludes this method at present.

Bryant and Flemming[33] report the use of veratrum viride in the treatment of eclampsia. This drug has been tried by many physicians in the past years. It was used at the Massachusetts Memorial Hospitals in 1912–1913 in doses up to 230 drops by mouth in eleven and a half hours and 360 drops subcutaneously in seventy-two hours, and it did appear to cause a drop in pulse rate; the blood-pressure recordings were inadequate. It was discontinued because it did not seem to give improved results. However, in those years, ob-

stetric treatment was radical, including *accouchement forcé*, and little or no treatment for shock; moreover, without intravenous magnesium sulfate therapy, it is difficult to see how any medical treatment could have had a fair trial. Workers at the Cincinnati Hospital, using veratrum viride, intravenous magnesium sulfate, concentrated glucose solutions, sedatives only for extreme restlessness and labor and, rarely, morphine, have treated 120 consecutive cases of eclampsia with 2 deaths, a mortality of less than 2 per cent, which is remarkable.

Veratrum viride is regarded as a powerful drug, and individual susceptibility to the drug is marked. It is described as being a vasodilator, and spasm of the blood vessels is therefore relieved and the blood supply to the tissues is increased.

It seems apparent that veratrum viride should be used with great caution. Until results are obtained from others who are now using the drug according to the routine of Bryant and Flemming,[33] it should not be routinely employed. If the mortality rate of eclampsia can be lowered in other hospitals, it is evident that veratrum viride has an important place in treatment.

Summary and Conclusions

Recent theories on the cause and results of eclampsia are reviewed. The physiologic and cellular pathology is discussed, the damage caused to the vascular system being stressed.

Statistics of 55 consecutive cases of eclampsia, with comparative statistics, are presented.

The treatment of eclampsia is outlined. Since the mortality rates show that treatment continues to be inadequate, conservative therapy should be employed until a better method can be substantiated.

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References

1. Hunt, H. F., Patterson, W. B., and Nicodemus, R. E. Placental infarction and eclampsia. *Am. J. Clin. Path.* **10**:319-331, 1940.
2. Patterson, W. B., Nicodemus, R. E., and Hunt, H. F. Hypothyroidism — etiologic factor in eclampsia. *Pennsylvania M. J.* **41**:983-988, 1938.
3. Bartholomew, R. A., and Kracke, R. R. The probable rôle of the hypercholesteremia of pregnancy in producing vascular changes in the placenta, predisposing to placental infarction and eclampsia. *Am. J. Obst. & Gynec.* **31**:549-562, 1936.
4. Peters, J. P., Lavieies, P. H., and Zimmerman, H. M. Pyelitis in toxemias of pregnancy. *Am. J. Obst. & Gynec.* **32**:911-927, 1936.
5. Mussey, R. D., and Lovelady, S. B. Is there a clinical relationship between pyelitis of pregnancy and pre-eclamptic toxemia? *Am. J. Obst. & Gynec.* **39**:236-242, 1940.
6. Prather, G. C., and Sewall, W. Relation of pyelonephritis to toxemias of pregnancy. *Surg., Gynec. & Obst.* **72**:781-786, 1941.
7. Strauss, M. B. The toxemias of pregnancy. *New York State J. Med.* **40**:810-818, 1940.
8. Smith, G. V., and Smith, O. W. Excessive gonad-stimulating hormone and subnormal amounts of oestrin in toxemias of late pregnancy. *Am. J. Physiol.* **107**:128-145, 1934.
9. Venning, E. H. Gravimetric method for the determination of sodium pregnandiol glucuronidate (an excretion product of progesterone). *J. Biol. Chem.* **119**:473-480, 1937.
10. Weil, P. G. The excretion of pregnandiol in the toxemias of pregnancy. *Science* **87**:72, 1938.
11. Smith, G. V., Smith, O. W., and Pincus, G. Total urinary estrogen, estrone and estriol during a menstrual cycle and pregnancy. *Am. J. Physiol.* **121**:98-106, 1938.
12. Pincus, G., and Zahl, P. A. The biogenesis of primary sex hormones. I. The fate of estrins injected into the rabbit. *J. Gen. Physiol.* **20**:879-893, 1937.
13. Evans, H. M., Kohls, C. L., and Wonder, D. H. Gonadotropic hormone in the blood and urine of early pregnancy: the normal occurrence of transient extremely high levels. *J. A. M. A.* **108**:287-289, 1937.
14. Smith, G. V., and Smith, O. W. Evidence for the placental origin of the excessive prolan of late pregnancy toxemia and eclampsia. *Surg., Gynec. & Obst.* **61**:175-183, 1935.
15. *Idem*. Estrogen and progestin metabolism in pregnancy; endocrine imbalance of pre-eclampsia and eclampsia: summary of findings to February, 1941. *J. Clin. Endocrinology* **1**:470-476, 1941.
16. Weiss, S., Dexter, L., Parker, F., Jr., and Tenney, B., Jr. Arterial hypertension in pregnancy and the hypertensive toxemia syndrome of pregnancy (preeclampsia and eclampsia). *Tr. A. Am. Physicians* **55**:282-288, 1940.
17. Dieckmann, W. J. The prevention and treatment of eclampsia. *Am. J. Surg.* **48**:101-111, 1940.
18. Irving, F. C. The vascular aspect of eclampsia. *Am. J. Obst. & Surg.* **31**:466-476, 1936.
19. Bell, E. T. Renal lesions in the toxemias of pregnancy. *Am. J. Path.* **8**:1-41, 1932.
20. Hertig, A. T. Personal communication.
21. Tenney, B., Jr., and Parker, F., Jr. The placenta in toxemia of pregnancy. *Am. J. Obst. & Gynec.* **39**:1000-1005, 1940.
22. Weintraub, F. A. Statistical review of eclampsia: based on twelve years' experience in Israel Zion Hospital. *New York State J. Med.* **40**:112-116, 1940.
23. Ware, H. H., Jr., and Noblin, F. E. A review of eclampsia at the Medical College of Virginia hospitals. *South. M. J.* **30**:153-158, 1937.
24. Teel, H. M., and Reid, D. E. Eclampsia and its sequelae: a clinical and follow-up study of all cases at Boston Lying-in Hospital over a twenty-year period. *Am. J. Obst. & Gynec.* **34**:12-25, 1937.
25. Torpin, R., and Coppedge, W. W. Eclampsia: review of three hundred and fifty cases stressing therapy. *South. M. J.* **33**:673-681, 1940.
26. Peckham, C. H. An analysis of 127 cases of eclampsia treated by the modified Stroganoff method. *Am. J. Obst. & Gynec.* **29**:27-35, 1935.
27. Chesley, L. C., and Somers, W. H. Eclampsia and posteclamptic hypertension: a follow-up study with an analysis of factors affecting the remote prognosis. *Surg., Gynec. & Obst.* **72**:872-888, 1941.
28. Rucker, M. P. The prognosis in eclampsia. *Virginia M. Monthly* **58**:369-376, 1931.
29. Lazard, E. M. An analysis of 575 cases of eclamptic and pre-eclamptic toxemias treated by intravenous injections of magnesium sulphate. *Am. J. Obst. & Gynec.* **26**:647-656, 1933.
30. Davis, A. B., and Harrar, J. A. Toxemia of pregnancy: eight hundred and seventy-nine cases with convulsions at the New York Lying-in Hospital. *J. A. M. A.* **87**:233-236, 1926.
31. Schwarz, O. H., and Dorsett, E. L. Further observations on the conservative treatment of eclampsia. *South. M. J.* **23**:288-293, 1930.
32. Kimbrough, R. A., and Shirey, R. M. A statistical survey of eclampsia: based on six years' experience in Philadelphia Lying-in Hospital. *Am. J. Obst. & Gynec.* **32**:415-420, 1936.
33. Bryant, R. D., and Flemming, J. G. Veratrum viride in the treatment of eclampsia: II. *J. A. M. A.* **115**:1333-1339, 1940.
34. Greenhill, J. P. Eclampsia at the Chicago Lying-in Hospital: immediate and late results. *J. A. M. A.* **87**:228-232, 1926.
35. Williams, W. *Obstetrics*. 1137 pp. New York: D. Appleton-Century Co., 1930.
36. Stander, H. J. Cited by Irving, F. C. *A Textbook of Obstetrics for Students and Practitioners*. 558 pp. New York: The Macmillan Co., 1936.
37. Chesley, L. C. Certain laboratory findings and interpretations in eclampsia. *Am. J. Obst. & Gynec.* **38**:430-437, 1939.
38. Cadden, J. F., and Stander, H. J. Uric acid metabolism in eclampsia. *Am. J. Obst. & Gynec.* **37**:37-47, 1939.
39. Stander, H. J., and Radelet, A. H. The blood chemistry in eclampsia. *Johns Hopkins Hosp. Bull.* **38**:423-430, 1936.
40. Arnold, J. O., and Fay, T. Eclampsia: its prevention and control by means of fluid limitation and dehydration. *Surg., Gynec. & Obst.* **55**:129-150, 1932.
41. May, G. E. Dehydration therapy in the toxemias of pregnancy: a report of sixty-five cases. *New Eng. J. Med.* **215**:277-289, 1936.
42. Davis, M. E. Water balance in relation to toxemias of pregnancy. *Surg., Gynec. & Obst.* **66**:426-431, 1938.
43. Dieckmann, W. J. Blood and plasma volume changes in eclampsia. *Am. J. Obst. & Gynec.* **32**:927-936, 1936.
44. Fischer, M. H. Diagnosis, prognosis and treatment in nephritis. *Detroit M. J.* **16**:227-260, 1916.
45. Dean, N. D. Bronchoscopy as an adjunct in the treatment of acute pulmonary edema complicating eclampsia. *Am. J. Obst. & Gynec.* **39**:886, 1940.
46. Smith, G. V. Personal communication.

DIABETES INSIPIDUS AND PREGNANCY*

Report of Two Cases

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DIABETES insipidus is comparatively rare, and the combination of this disease and pregnancy is still rarer. It is seldom met with in practical obstetrics, and since 1915 there have been only 3 such cases in approximately 50,000 deliveries at the Boston Lying-in Hospital. From the scarcity of reported cases of the combination of pregnancy and diabetes insipidus, the impression is obtained that very few patients with this disease have children. However, in a thorough study of the literature, it was found that patients with diabetes insipidus have children more frequently than one would expect.[1] At times they are quite prolific, some having as many as eleven children during the course of the disease.

One of the most interesting cases to illustrate this point was reported by Bellot and Brougniart[2] to the Philomatical Society on October 22, 1791. The patient was a forty-year-old Frenchwoman, who was married at the age of twenty-two. She had had a very considerable thirst from her earliest infancy. While she was single, she drank three pailfuls of water a day. After she was married, two pailfuls were sufficient until she was delivered of her first child, when she returned to her former quantity of three pailfuls and continued it until after the birth of her fourth child. After that period, she drank only two pailfuls in twenty-four hours. When she was pregnant, she had much more thirst than usual. She had eleven children in ten pregnancies. In the presence of a commission, this woman drank 14 liters of water during the space of ten hours. One of Gee's[3] patients had nine children. Weil[4] and his son[5] studied five generations of a family of 220 members, headed by a man with diabetes insipidus, of whom 35 had the disease. Some of the women with diabetes insipidus had eight or nine children, with normal pregnancies and birth, except that the pregnancies usually increased the symptoms of the disease.

On the basis of a perusal of the literature, the clinical effects of the combination of diabetes insipidus and pregnancy may be classified as follows: aggravation of the disease during pregnancy; transitory diabetes insipidus during pregnancy; amelioration of the disease during pregnancy; no effect of pregnancy on the disease; onset of the disease during pregnancy and persistence after delivery; and transient appearance of the disease after delivery.

Aggravation of Diabetes Insipidus during Pregnancy

A number of reports indicate that the polyuria and polydipsia of diabetes insipidus may be increased by varying degrees and during various periods of pregnancy,[6–10] as already mentioned. Among 26 members with diabetes insipidus in a family of 73, Ellerman[10] found increased diuresis during pregnancy of the women with diabetes insipidus. In 1 patient, who gave birth eight times, the diuresis increased during each pregnancy from around 10 to approximately 20 liters daily; during the seventh pregnancy, the daily quantity of urine reached 30 liters.

Transient Diabetes Insipidus during Pregnancy

The greatest number of reports on any one of these phases of diabetes insipidus and pregnancy appear in connection with the transient occurrence of the disease during a normal pregnancy.[11–29] The disease may occur in an apparently normal woman during any stage of pregnancy, from the beginning to near the end of term. However, in most cases, the diabetes insipidus was apparent in the latter half of pregnancy, usually in the fifth or sixth month. Ordinarily, the polyuria and the polydipsia abruptly ceased two or three days after delivery, although in some cases it persisted for as long as seventeen days post partum. There have been cases in which the transient diabetes insipidus occurred in successive pregnancies, in approximately the same months of gestation. In most of these cases, the fluid intake and output increased to about 10 or 12 liters. However, in Edelmann and Kritzman's[22] case, the daily urine volume reached a level of 25 liters. Novak's[17] case was of interest because the diabetes insipidus appeared in the course of the first pregnancy and disappeared after delivery; this phenomenon reappeared in the course of ten pregnancies. Gänsslen and Fritz[20] presented an unusually interesting case of a healthy woman whose husband had diabetes insipidus. During the terminal part of her first pregnancy, she showed symptoms, which disappeared after delivery. The baby of this pregnancy subsequently had diabetes insipidus. During her second preg-

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nancy, which resulted in a healthy baby, the patient had no polyuria. The authors suggested the possibility that the hormone of the sick child was transferred to the mother during the terminal part of pregnancy and gave her the symptoms of the disease.

Amelioration of Diabetes Insipidus during Pregnancy

Several reports have shown that pregnancy occurring during the course of diabetes insipidus resulted in an amelioration of the disease, and after delivery the polyuria and the polydipsia reappeared abruptly.[9, 12, 16, 30–34] The most interesting report on this aspect of the problem is that given by Duvoir, Pollet and Cachin,[33] who treated a woman with diabetes insipidus during the course of three pregnancies and observed that the polyuria decreased in the sixth month of each and that the amount of urine became normal during the weeks preceding delivery. However, on the day following delivery, the daily urine increased suddenly to 12 liters, the level before pregnancy. Carter[34] noted that a patient had less thirst in the seventh month of pregnancy, and that after a premature delivery, the diabetes insipidus was considerably relieved and remained so during the years of subsequent observation.

No Effect of Pregnancy on Diabetes Insipidus

A number of studies show that pregnancy had no effect on the polyuria and the polydipsia of diabetes insipidus.[31, 35–40, 65] Some of these patients had urine volumes as high as 16 to 20 liters a day. The patients of Boissard[35] and Artaud[38] excreted similar amounts of urine without any alteration during pregnancy.

Onset of Diabetes Insipidus during Pregnancy

French[31] observed a woman who developed diabetes insipidus with a daily urine volume of 10 liters during the fourth month of her fifth pregnancy. The polyuria and polydipsia persisted after delivery. Vickers[41] treated a patient who developed diabetes insipidus with an output of approximately 8 liters when she was three months pregnant. She had urinary retention and, as a result, had to be catheterized; 3250 cc. of urine was obtained. She miscarried a normal three-month-old fetus, and the diabetes insipidus persisted after the miscarriage.

Transient Diabetes Insipidus after Delivery

Transient diabetes insipidus may also appear shortly after delivery. Laurentie and Basiliou[42] presented the case of a thirty-seven-year-old woman who went through a normal pregnancy. On the day after delivery, the patient suddenly began to void frequently and to drink with a "rage." Twenty-one days later, she developed violent frontal headaches, photophobia and convulsions. In addition, a transient hypertension appeared. The whole picture was typical of eclampsia; however, there was no albumin in the urine. The fluid intake was as high as 15 liters a day. At one time, the patient had to be catheterized because of a dilated bladder, and 3 liters of urine was obtained. Within six weeks after the onset of convulsions, all symptoms disappeared, and the patient was discharged from the hospital completely recovered.

There are a number of miscellaneous reports on pregnancy and diabetes insipidus, although many of them do not add individual case reports.[43–53] It appears, however, that the various authors have had experience on this subject. Theses have been published by Jansen[46] and Schellenberg.[47] Jansen added a case of a woman who had six deliveries, but the diabetes insipidus appeared only during the last three pregnancies and lasted, each time, until fourteen days post partum. Discussions on this problem have been presented by Lauter and Hiller,[48] Skrobanski,[49] Kleinwächter[50] and Henriet.[51]

Case Reports

Case 1. A 28-year-old woman, who had had diabetes insipidus for over 9 years, had been observed by one of us (H. B.) for 8 years, during which various data were obtained. The daily fluid intake and urine volume usually varied from 10 to 12 liters. The past and family histories were irrelevant. The menses were normal, except that the intramuscular injection of pituitary extract caused painful menses. General physical examinations were normal, except that the patient was about 15 pounds under weight.

The basal metabolic rate was −18 per cent. Lumbar puncture showed a normal spinal fluid. The blood Wassermann reaction was negative. X-ray examination of the skull was negative. The patient required 1 cc. of pituitary extract intramuscularly three or four times a day to reduce her fluid intake and output to about 3 liters. In the preceding 2 years, she had taken powdered pituitary extract intranasally five or six times a day, with relief. Blood studies made before pregnancy were essentially negative, both with and without pituitary extract; some of the results are shown in Table 1.

In 1936, because of various reports suggesting that the administration of estrogenic substance may suppress the diuretic principle of the anterior pituitary gland,[54] the patient was injected with 20,000 units of amniotin daily for 10 days, without any effect on the fluid intake and output.

The patient had her last menstrual period on June 24, 1940. Her expected delivery date was March 31, 1941. Physical examination was negative except that she weighed only 100 pounds, which was low for her height of 65 inches. The pregnancy progressed normally for both patient and fetus. She had no nausea or vomiting at any time and felt just as well as when she was not pregnant. During the beginning of pregnancy, the daily fluid intake and output were about 10 to 12 liters, the level before

conception, and she took powdered pituitary extract intranasally, with a reduction in these volumes. She felt movements of the fetus in the 5th month. The blood pressure remained constant at approximately 120/70. X-ray films of the pelvis taken on December 31, at the end of the 6th month of pregnancy, showed a fetus consistent with the date.

The daily fluid intake and output are of unusual interest because they increased to a level of about 13 to 15 liters at the end of the 7th month of pregnancy. When the patient was 7½ months pregnant, the polyuria and polydipsia began to decrease appreciably, so that shortly before delivery the daily fluid intake and output were about 3 liters for some days (Fig. 1). She went through a normal labor and was delivered of a normal baby on March 28. The convalescence was normal. The day after delivery, the fluid intake and output increased abruptly to a level of 8 liters, and remained at approximately that level during the subsequent 9 months of observation. These volumes were somewhat lower than those noted before pregnancy. The patient had no edema at any time during or after pregnancy.

It was interesting that during pregnancy the administration of pituitary extract caused a sensation of constriction of the uterus and that, as a result, the extract was administered only in small doses. However, when Pitressin, which is supposedly pituitary extract without the oxytocic principle, was administered, it had an antidiuretic effect, without the constrictive action on the uterus. This was shown experimentally by the use of a tocograph, which, when properly placed on the abdomen, mechanically records and times the uterine contractions. No uterine contractions were elicited when 0.5 cc. of Pitressin was injected subcutaneously. However, normal and rhythmic uterine contractions were obtained when 0.5 cc. of pituitary extract was injected.

The placenta weighed 840 gm. and measured 23 by 20 by 2.5 cm. The membranes were complete. The cord was attached 2 cm. from the nearest placental margin, measured 45 by 1 cm., and was normal on section. The fetal surface was shiny, bluish gray. The maternal surface was intact and very fissured, deep reddish gray and soft. The cut surface was deeply congested and soft. Microscopically, there was a moth-eaten and fibrinoid degeneration of the decidua. There were foci of recent hemorrhage on the decidua. The villi were slightly larger than normal ones at full term but were well vascularized. The syncytial nuclear knots were infrequent. The stroma was not remarkable. The chorion was normal. The placenta appeared normally mature.

At birth, the baby's weight was 8¼ pounds, and its height 55 cm. There was an initial loss of 12 ounces, but the birth weight was regained on the 10th day. The baby was breast fed for 31 days. There was an adequate milk supply during this time, but subsequently the mother's breasts dried up rapidly and the baby was given a milk formula. At the age of 6 months, the baby weighed 16 pounds and had one tooth. At 8 months, his weight was 20¼ pounds, and his height 74 cm. He had been perfectly normal and had taken a normal amount of liquids.

Various laboratory data were obtained on this patient before, during and after pregnancy (Table 1). The results of the blood volumes were approximately the same as those found during and after pregnancy in the normal woman.[55] In the terminal part of pregnancy, the hematocrit showed a decreased percentage of corpuscles, which increased to 34 per cent shortly after delivery. This compared with 35 per cent obtained a number of times before pregnancy. The venous pressure was equivalent to 10 cm. of water. The circulation time was 14 seconds by the cyanide method. The basal metabolic rate increased toward the end of the pregnancy to +1 per cent, but within 10 days after delivery, it had decreased to −19 per cent, the level before pregnancy.

Sugar-tolerance tests were made during and shortly after pregnancy, and these results were much the same. However, the blood-sugar curves were lower than those obtained several years before pregnancy, as shown in Table 2.

TABLE 1. *Laboratory Data before, during and after Pregnancy.*

DATE	WEIGHT	BLOOD VOLUME			HEMATOCRIT		SERUM PROTEIN	CHOLESTEROL	BASAL METABOLIC RATE	PITUITARY EXTRACT GIVEN	FLUID INTAKE
		TOTAL VOLUME	PLASMA VOLUME	CORPUSCULAR VOLUME	CORPUSCLES	PLASMA					
	lb.	*cc.*	*cc.*	*cc.*	%	%	*gm./100 cc.*	*mg./100 cc.*	%		*liter.*
Before pregnancy											
Without therapy	100	—	—	—	35	65	7.0	144	−17	No	11.0
With therapy					35	65	6.4	154		Yes	3.0
During pregnancy											
1/30/41	111	4630	3195	1435	31	69	6.4		−5	No	13.0
2/7/41	113	4950	3460	1490	30	70	6.3		−11	Yes	5.5
3/7/41	113	4810	3360	1450	30	70	6.1	225	−5	No	4.1
3/9/41	115	4800	3450	1350	28	72	6.1	215	+1	Yes	2.7
After delivery											
4/8/41	101	3485	2300	1185	34	66			−19	No	7.5
4/10/41	101	3460	2300	1160	34	66			−20	Yes	2.8

TABLE 2. *Sugar-Tolerance Tests before, during and after Pregnancy.*

DATE	BLOOD-SUGAR LEVELS					PITUITARY EXTRACT GIVEN
	FASTING	½ HR.	1 HR.	2 HR.	3 HR.	
	mg./100 cc.	*mg./100 cc.*	*mg./100 cc.*	*mg./100 cc.*	*mg./100 cc.*	
Before pregnancy						
4/7/33	66	314	—	159	54	
4/11/33	61	196	214	133	52	No
During pregnancy						
3/8/41	100	167	172	135	116	No
3/11/41	95	147	167	127	122	Yes
After delivery						
4/7/41	105	145	178	135	116	No
4/9/41	131	145	167	153	145	Yes

Case 2. A 36-year-old woman was seen in consultation by one of us (H. B.) with Dr. Henry S. Finkel, who treated her during her pregnancies. The family, past and menstrual histories were irrelevant. In 1936, she was delivered of a female child by low cervical cesarean section after a long test of labor, because of cephalopelvic disproportion. There was no polyuria or polydipsia at that time or immediately thereafter.

In the present pregnancy, the last menstrual period began on August 17, 1941. The expected date of confinement was May 24, 1942. General physical examination and routine blood and urine tests were negative. The height was 64 inches, and the weight was 140 pounds. The pregnancy progressed normally until about the 5th month, when the patient began to notice polydipsia and polyuria, which increased progressively as the pregnancy advanced. There was no unusual weight gain. During the last 2 months of pregnancy, the average daily fluid intake and output were about 8000 cc., with maximum and minimum levels of 9200 and 7600 cc. The urine had a specific gravity of 1.002.

On April 27, 1942, x-ray examination revealed a twin pregnancy. Because of the excessive size of the abdomen and the extreme discomfort of the patient, she was delivered prematurely on May 6, 18 days before term, by a low cervical cesarean section under spinal anesthesia. Normal twin boys weighing 5¾ pounds and 7⅛ pounds, respectively, were delivered. There was a double-ovum, twin, fused placenta.

The convalescence was uneventful except for considerable abdominal distention for the first few days. The polydipsia and polyuria disappeared abruptly after the delivery. The fluid intake and output were normal during the subsequent 3 months of observation. The specific gravity of the urine increased to about 1.015.

Discussion

A review of the literature indicates that pregnancy may aggravate, ameliorate or not influence diabetes insipidus. In addition, the disease may appear during any stage of pregnancy, disappearing or persisting after delivery. In the first case presented, the polyuria and polydipsia showed some increase in the seventh month of pregnancy and a gradual reduction during the eighth month, so that for some days before delivery the fluid intake and output were about 3 liters. After a normal delivery of a normal baby, the polyuria and polydipsia suddenly rose to about 8 liters, which was somewhat lower than the level before conception took place. In the second case, transient diabetes insipidus appeared in a normal woman in her fifth month of pregnancy and disappeared suddenly after delivery by cesarean operation.

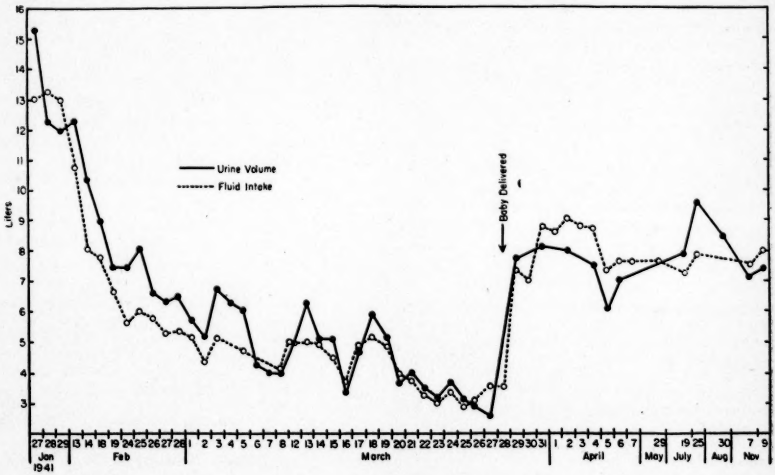

Figure 1.

This chart records the daily fluid intake and urinary output during the last two months of pregnancy and after delivery of the patient in Case 1; during this period, no pituitary extract was administered.

The cause for these variable effects of pregnancy on diabetes insipidus is conjectural. The excretion of water is controlled by a balance of the hormones that diminish and those that produce diuresis. Dexter and Weiss[56] believe that the mechanisms of oliguria of pregnancy are not uniform in the absence of cardiac or renal disease. The variable effects apparently depend on which of these hormonal forces predominates. It is suggested that

the diuretic or antidiuretic hormone of the fetus may be transferred to some mothers and produce the effects of each in the mother. In the first case presented above, a marked improvement in the diabetes insipidus toward the end of pregnancy was conceivably due to the antidiuretic action of the posterior pituitary lobe of the fetus or to the increased activity of that of the mother. On the other hand, the patient of Gänsslen and Fritz[20] suggests the possibility that the diuretic hormone of the fetus was transferred to the mother, with the resultant temporary symptoms of diabetes insipidus.

The hormones that diminish diuresis include the hormone of the posterior pituitary lobe. Its value is well established. In addition, follicular hormone, which is normally increased during pregnancy, has been used with success in the treatment of polyuria without pregnancy by Troisier[57] and Beltrametti,[58] although this effect was not observed by Blotner.[54] The former injected large doses of folliculin into patients with diabetes insipidus and obtained a marked antidiuretic effect. In the first case presented above, no such effect was obtained before pregnancy. Müller[30] referred to Scanzoni, who observed a nonpregnant woman with sparse menses and polyuria and noted that when the menses became regular the polyuria diminished.

The diuretic hormones are most probably of anterior pituitary or placental origin. Henriet[51] referred to Lévy-Solal, who presented a polyuric patient with a hydatidiform mole, a condition in which the urinary prolan is markedly increased. In addition, Lévy-Solal observed a pregnant woman who, in spite of edema, had a significant polyuria. The determination of her daily prolan excretion gave the enormous figure of 38,000 rabbit units.

The diuretic action of the thyroid hormone is also known. However, in the first case reported above, the thyroid gland probably had no effect on the polyuria because the metabolism increased from −18 to +1 per cent when the urinary volume had dropped to 3 liters a day. After delivery, the patient's metabolism decreased to −19 per cent, and polyuria again developed. The combination of these findings is contrary to those observed by Blotner and Cutler[59] in patients with diabetes insipidus whose polyuria was relieved by total thyroidectomy.

The blood volumes determined with Evans blue[60, 61] were of interest because they were normal in spite of the interchange of huge volumes of fluids. The administration of pituitary extract did not dilute the blood or change significantly the blood volumes either during or after pregnancy. These results are in agreement with blood volumes determined by Blotner[62] with Congo red[63] in 2 male patients with diabetes insipidus, and also those determined with Evans blue by Gibson[60] in 3 similar patients. They showed no significant changes with the administration of pituitary extract. Soule [39] also found the blood volume to be within normal physiologic limits in a patient with diabetes insipidus who was about four and a half months pregnant.

The duration of labor and the birth of children in patients with diabetes insipidus are normal and spontaneous. However, there are rare patients with premature delivery, in striking contrast to the parturition in cats with experimental diabetes insipidus, which were totally or partially unable to deliver their young or had prolonged labor.[64] Fisher, Magoun and Ranson[64] believed this disturbance to be due to a deficiency in the pituitary oxytocic hormone consequent to the experimental diabetes insipidus.

Lactation, as it appeared in our cases and as it is reported in the literature, is much like that after birth in the normal woman. In some cases, there is an adequate milk supply, and in others practically no milk.

In the treatment of a pregnant patient with diabetes insipidus, care must be taken about the use of pituitary extract because of its effect on the uterus. If pituitary extract is used, small doses, such as 1 to 3 minims, may be injected. The intranasal route is advantageous because it eliminates the marked effect on the uterus noticed with injection. However, intranasal application does not appear so effective during pregnancy as in the nonpregnant state. Pitressin appears more valuable than pituitary extract because it lacks the oxytocic principle.

Summary

A review of the literature and the details of two cases of diabetes insipidus and pregnancy are presented. The first patient went through a normal pregnancy and gave birth to a normal child. The fluid intake and output increased when she was seven months pregnant and then gradually decreased to near normal shortly before confinement. One day post partum, the polyuria and polydipsia abruptly returned. The second patient, in the fifth month of pregnancy, developed transient diabetes insipidus, which disappeared suddenly after she was delivered of normal twins eighteen days before term by cesarean section.

Existing diabetes insipidus may be intensified, improved or unaltered by pregnancy; the disease may also appear during any stage of gravidity, and may cease rapidly or persist after delivery.

References

1. Blotner, H. The inheritance of diabetes insipidus. *Am. J. M. Sc.* **204**:261-265, 1942.
2. Bellot, M. M., and Brougniart. Report made to the Philomatical Society,relative to a woman who drinks a great quantity of water. In *Simmons's Medical Facts and Observations*. Vol. 2. London: J. Johnson, 1792. P. 87.
3. Gee, S. A contribution to the history of polydipsia. *St. Barth. Hosp. Rep.* **13**:79, 1877.
4. Weil, Adolf. Über die hereditäre Form des Diabetes insipidus. *Virchows Arch. f. path. Anat.* **95**:70-96, 1884.
5. Weil, Alfred. Über die hereditäre Form des Diabetes insipidus. *Deutsches Arch. f. klin. Med.* **93**:180-200, 1908.
6. Chavane, and Faure-Miller, R. Diabète insipide et grossesse. *Bull. Soc. d'obst. et de gynéc.* **3**:400-409, 1900.
7. Ballerini. Per la genesi del diabete insipido e delle poliurie gravidiche. *Folia Gynaec.* **14**:97, 1921.
8. Klaften, E. Diabetes insipidus und Schwangerschaft. *Monatschr. f. Geburtsh. u. Gynäk.* **75**:333-349, 1927.
9. Momigliano, E. Diabete insipido e gravidanza. *Ann. di ostet. e ginec.* **51**:905-979, 1929.
10. Ellerman, M. Le diabète insipide héréditaire. *Acta psychiat. et neurol.* **14**:233-241, 1939.
11. Bennett, J. H. *Clinical Lectures on the Principles and Practice of Medicine*. Fifth edition. 1037 pp. Edinburgh, 1868.
12. Duncan, J. M. On diabetes insipidus in pregnancy and labour. *Tr. Obst. Soc. London* **29**:308-316, 1888.
13. Shamberg, J. F. A case of exaggerated polyuria of pregnancy. *Univ. Mag.* **6**:385, 1893-94.
14. Voituriez, J. De la polyurie gravidique. *J. de Soc. méd. de Lilles* **1**:457-467, 1890.
15. Esterle. Cited by Tarnier, S., and Budin, P. *Traité de l'art des accouchements*. Vol. 2. 586 pp. Paris: G. Steinheil, Libraire-Éditeur, 1886.
16. Vinay, C. Diabète insipide et grossesse. *Semaine méd.* **19**:14, 1899.
17. Novak, J. Diabetes insipidus in graviditate. *Berl. Klin. Wchnschr.* **54**:107-109, 1917.
18. Lichwitz, L. Drei Fälle von Simmondscher Krankheit. *Klin. Wchnschr.* **1**:1877-1879, 1922.
19. Heidler, H. Kongenitale Hautdefekte bei Neugeborenen. *Zentralbl. f. Gynäk.* **48**:357-363, 1924.
20. Gänsslen, M., and Fritz. Über Diabetes insipidus. *Klin. Wchnschr.* **3**:22, 1924.
21. Frühinsholz, A., Vermelin, H., and Hennequin. Diabète d'origine hypophysaire et gestation. *Bull. Soc. d'obst. et gynéc.* **17**:741-744, 1928.
22. Edelmann, J. G., and Kritzman, A. S. Ein Fall von Diabetes insipidus bei einer Schwangeren mit Lues ignorata. *Ber. ü. d. ges. Gynäk. u. Geburtsh.* **10**:325, 1926.
23. Mestitz, W. Ein Fall von transitorischer Polyurie und Polydipsie am normalen Schwangerschaftsende. *Ztschr. f. Geburtsh. u. Gynäk.* **98**:123-129, 1930.
24. Anselmino, K. J., and Hoffmann, F. Diabetes insipidus und Schwangerschaft. *Zentralbl. f. Gynäk.* **54**:2061-2068, 1930.
25. Dietel, H. Diabetes insipidus und Schwangerschaft. *Arch. f. Gynäk.* **159**:404-409, 1935.
26. Brattström, E. Ein Fall von Diabetes insipidus traumaticus und Gravidität. *Acta obst. et gynec. Scandinav.* **18**:320-324, 1938.
27. Bleakly, J. Case of diabetes insipidus and twin pregnancy. *Proc. Roy. Soc. Med.* **31**:1062-1064, 1938.
28. Merlino, A. Diabete insipido complicante le gravidanza. *Arch. di ostet. e ginec.* **2**:121-143, 1938.
29. Hart, S. D., and Breitman, H. B. Diabetes insipidus complicating pregnancy. *Am. J. Obst. & Gynec.* **41**:527, 1941.
30. Merbsch. Cited by Müller, P. *Die Krankheiten des weiblichen Körpers in ihren Wechselbeziehungen zu den Geschlechtsfunctionen*. 508 pp. Stuttgart, 1888.
31. French, H. The influence of pregnancy upon certain medical diseases, and of certain medical diseases upon pregnancy. *Brit. M. J.* **1**:1165-1172, 1908.
32. Katz, H. Cited by Heidler.[10] *Zentralbl. f. Gynäk.* **48**:357-363, 1924.
33. Duvoir, M., Pollet, L., and Cachin, M. Diabète insipide suivi de diabète sucré avec coma: influence des grossesses sur la polyurie: traitement par la folliculine. *Bull. et mém. Soc. méd. d. hôp. de Paris* **48**:1444-1450, 1932.
34. Carter, P. J. Diabetes insipidus in pregnancy: case report. *Urol. & Cutan. Rev.* **44**:549-551, 1940.
35. Boissard. Discussion of Chavane and Faure-Miller.[6]
36. Bar. Discussion of Chavane and Faure-Miller.[6]
37. Kölle. Demonstration: Gravidität bei Diabetes insipidus. *Zentralbl. f. Gynäk.* **50**:2212, 1926.
38. Artaud, A. Un cas de grossese au cours d'un diabète insipide. *Bull. Soc. d'obst. et de gynéc.* **22**:196-198, 1933.
39. Soule, S. D. Diabetes insipidus and pregnancy. *Am. J. Obst. & Gynec.* **33**:878-890, 1937.
40. Verdeuil, and Casalta. Une observation de diabète insipide et gravidite. *Bull. Soc. d'obst. et de gynéc.* **27**:87, 1938.
41. Vickers, D. M. Diabetes insipidus with acute retention in pregnancy: report of a case. *Surg., Gynec. & Obst.* **38**:223-225, 1924.
42. Laurentie, A., and Basiliou, B. J. Crises éclamptiques sans albuminurie ayant apparu le 22e jour des suites de couches amaurose et diabete insipide: contribution a l'étude pathogénique du diabète insipide dans ses rapports avec l'eclampsie. *Gynec. et obst.* **13**:321-331, 1926.
43. Felletär, J. Diabetes insipidus and pregnancy. *Orvosi hetil.* **73**:701, 1929.
44. Marañon, G. Un caso de diabetes insípida gravídica con poliuria enorme y probable lesión tuberculosa de lóbula posterior de hipófisis. *Arch. Med. Madr.* **22**:289, 1926.
45. Pribrsky, J. Diabetes insipidus and pregnancy. *Časop. lék. cesk.* **73**:260, 299, and 322, 1934.
46. Jansen, R. *Über Diabetes insipidus (ein besonderer Fall)*. Inaugural Dissertation. Halle: C. A. Kaemmerer & Co., 1899.
47. Schellenberg, P. *Diabetes insipidus und Graviditat: ein Beitrag zur Aetiologie des Diabetes insipidus*. Inaugural Dissertation. 61 pp. Königsberg I: Johan Raabe, 1930.
48. Lauter, S., and Hiller, F. Diabetes mellitus und Diabetes insipidus in Auschluss an Schwangerschaft. *Deutsches Arch. f. klin. Med.* **146**:355-361, 1925.
49. Skrobanski, K. K. Über das Verhältnis des Diabetes mellitus und insipidus zu den weiblichen Geschlechtsorganen und Schwangerschaft. *Zentralbl. f. Gynäk.* **26**:1038, 1902.
50. Kleinwächter, L. Der Diabetes, vom gynäkologischen Standpunkt aus betrachtet. *Ztschr. f. Geburtsh. u. Gynäk.* **38**:191-233, 1898.
51. Henriet, P. Glycosuries et diabètes en pratique obstétricale. *Rev. franç. de gynéc. et d'obst.* **31**:111-128, 1936.
52. Silva, G. Diabetes insipidus und Schwangerschaft. *Bol. an. clin. Obst. Santiago del Chile* **14**:77, 1927.
53. Dellepiane, G. Sul diabete insipido complicante la gravidanza. *Boll. d. Soc. piemontese di ostet. de ginec.* **2**:429-450, 1934.
54. Blotner, H. Effect of amniotin and antuitrin-S in diabetes insipidus. *New Eng. J. Med.* **217**:592-594, 1937.
55. Thomson, K. J., Hirsheimer, A., Gibson, J. G., Jr., and Evans, W. A., Jr. Studies on circulation in pregnancy. III. Blood volume changes in normal pregnant women. *Am. J. Obst. & Gynec.* **36**:48-59, 1938.
56. Dexter, L., and Weiss, S. *Preeclamptic and Eclamptic Toxemia of Pregnancy*. 415 pp. Boston: Little, Brown and Company, 1941.
57. Troisier, J. Action antipolyurique des hautes doses de folliculine dans le diabète insipide de l'homme. *Bull. et mém. Soc. méd. d. hop. de Paris* **48**:1451-1455, 1932.
58. Beltrametti, L. Die Sexual funktionen in ihren Beziehungen zum Wasserhaushalt insbesondere zum Diabetes insipidus. *Endokrinologie* **16**:241-256, 1935.
59. Blotner, H., and Cutler, E. C. Total thyroidectomy in the treatment of diabetes insipidus. *J. A. M. A.* **116**:2739-2745, 1941.
60. Gibson, J. G., Jr., and Evans, W. A., Jr. Clinical studies of the blood volume. I. Clinical application of a method employing the azo dye "Evans blue" and the spectrophotometer. *J. Clin. Investigation* **16**:301-316, 1937.
61. Gibson, J. G., Jr., and Evelyn, K. A. Clinical studies of the blood volume. II. Adaptation of the method to the photoelectric microcolorimeter. *J. Clin. Investigation* **17**:153-158, 1938.
62. Blotner, H. Unpublished data.
63. Keith, N. M., Rowntree, L. G., and Geraghty, J. T. A method for the determination of plasma and blood volume. *Arch. Int. Med.* **16**:547-576, 1915.
64. Fisher, C., Magoun, H. W., and Ranson, S. W. Dystocia in diabetes insipidus: the relation of pituitary oxytocin to parturition. *Am. J. Obst. & Gynec.* **36**:1-9, 1938.
65. Riely, L. A. Personal communication.

MEDICAL PROGRESS

MILITARY DERMATOLOGY

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MILITARY dermatology concerns all cutaneous manifestations that are presented to medical officers under war conditions. Wherever medical officers may be ordered for duty over the present and future world-wide battlefronts, they cannot avoid being presented with cutaneous problems. Not all these physicians will have dermatologists available for consultation, and each officer will often be compelled to decide independently on these problems in military dermatology. Many of the diseases are infections caused by animal and vegetable parasites and the pyogenic organisms. The general principles of preventive medicine apply particularly to this group of cutaneous diseases. The medical officer has as his patients healthy young adults. He has the organization and discipline at hand for their control. His duty is to lessen disability and prevent "days lost" from active duty. Detection of disease and the correct diagnosis are the first necessity. With this knowledge, steps may be taken to prevent the disease and to avert its extension on the surface of the patient, its appearance in others and its complications. In the prevention of complications, attention should be given to avoiding not only the complications of the disease but also those of therapy — a significant point in the treatment of cutaneous disease. In the field of cutaneous medicine, as well as in other diseases in wartime, prophylactic measures are essential.

That this prevention is a necessary and vital factor is indicated by a review of the report on skin diseases published by the Surgeon General's office in 1928. This report[1] begins as follows:

> Diseases of the skin, exclusive of dermatological manifestations of venereal disease, though ordinarily considered to be of minor importance in so far as danger to life is concerned, are of great importance to an army operating in the field by reason of the noneffectiveness they cause. This was true of past wars and equally so of the World War.

There is no reason to expect that this statement will not be true in the present war, although a full utilization of the knowledge of cutaneous disease, if such knowledge could be applied, would certainly lessen the duration of disability and the incidence of disease.

The report summarizes the dermatologic figures for the Civil War as follows: itch, 35,236; skin diseases, 38,946; a total of 74,182 cases. Perhaps only these two skin diseases were recognized eighty-two years ago. In the statistics for World War I, the report includes relatively few cutaneous disorders, only about 126,000 cases being listed (Table 1). However, a review of this list indicates very clearly that many cases could not have been reported and that numerous skin diseases are not discussed. Fungus infections, for example, account for only 2000 cases, and trench feet, in another portion of the volume, for only 2000 cases. There were 3500 cases of ectoparasitism, and yet at the time of the Armistice, pediculosis probably involved 90 per cent of the men in the American Expeditionary Force. Little mention is made of the numerous secondary infections that probably occurred as a result of this infestation. A separate table indicates 18,000 cases in which skin disease was found concurrent with other disease for which hospital admission was made (Table 2). In addition, such diseases as erysipelas, anthrax, trench feet, mycoses, cancer of the skin, purpura and nail defects are not included in the special list of skin diseases.

TABLE 1. *Diseases of the Skin and Cellular Tissue Resulting in Primary Admission to All United States Army Hospitals, April 1, 1917, to December 31, 1919.*[1]

DISEASE	NO. OF CASES
Carbuncle	2,330
Furuncle	19,958
Abscess	16,329
Cellulitis	12,824
Trichophytosis	2,813
Ectoparasitism	3,269
Dermatitis	858
Eczema	4,035
Erythema	1,495
Herpes	3,141
Impetigo	2,735
Lichen	89
Pityriasis	579
Psoriasis	1,506
Scabies	34,134
Other diseases of skin and cellular tissue	20,270
Total	126,365

The statement quoted above regarding noneffectiveness is borne out by the number of days lost during the war (Table 3). The figures include

Reprints of articles in this series are not available for distribution, but the articles will be published in book form. The current volume is *Medical Progress: Annual, Vol. III, 1942* (Springfield, Illinois: Charles C Thomas Company, 1942. $5.00).

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only the diseases mentioned in the portion of the report entitled "Skin Diseases," omitting the cases found concurrently or those in the list of general diseases. It is obvious, therefore, that over 2,000,000 days of service were lost by reason of cutaneous affections to the Army alone during World War I.

Syphilis is also an incapacitating disease, this report[3] stating that there were 67,026 admissions for syphilis and 9665 other cases in which syphilis was found when the soldiers were admitted for other reasons. It is of interest that the admissions for syphilis represented only 1.85 per cent of the admissions to hospitals, and yet the total number of days lost from duty was over 1,927,201, and 143 deaths were charged to syphilis.

TABLE 2. *Concurrent Diseases of Skin and Cellular Tissue Found in Cases Admitted for Other Diseases to All United States Army Hospitals, April 1, 1917, to December 31, 1919.*[1]

DISEASE	NO. OF CASES
Carbuncle	158
Furuncle	1,763
Abscess	3,681
Cellulitis	1,718
Trichophytosis	411
Ectoparasitism	761
Dermatitis	150
Eczema	663
Erythema	352
Herpes	572
Impetigo	328
Lichen	19
Pityriasis	238
Psoriasis	356
Scabies	2,926
Other diseases of skin and cellular tissue	4,911
Total	19,007

The incidence of skin diseases in the Navy confirms the gravity of these diseases in a military organization. In a report by Sulzberger, MacClatchie and Parsons[4] citing statistics of the Medical Corps of the United States Navy, 14.8 per cent of all admissions for disease between 1929 and 1938 included men with skin diseases and syphilis. It is probable that a number of skin cases were omitted from these figures, which do not include, of course, the ambulatory patients. One and a quarter million days in hospitals were lost on account of these diseases. Skin diseases represented the third commonest cause of admission in this period, with a rate of 22.6 per thousand men, acute catarrhal fever ranking highest and gonorrhea second. These are peacetime figures and do not consider the inevitable increase of certain parasitic infections in time of war. Of individual diseases, scabies had the highest admission rate and trichophytosis next, and trichophytosis ranked highest in number of sick days per year per thousand men.

In addition, a personal letter from a dermatologist at one of the naval stations shows that 13 per cent of the total admissions to the hospital in the first eight months of its existence were on the dermatologic service. This figure includes all the common diseases of the skin in stages that rendered the service men temporarily unfit for duty. Thirty per cent of these admissions were for fungus infections of the skin, and 20 per cent were for scabies. It is stated that preventive methods are in force at this particular station, and yet these admissions continue. It is furthermore of interest that almost all the patients admitted to the hospital had already been overtreated in their particular outfits. This comment of overtreatment has come to me from several other sources. The complication may occur because of the failure to make a proper diagnosis or to estimate the tolerance of an individual skin, or because of the desire to clear up the existing skin trouble quickly by strong measures.

TABLE 3. *Days Lost from Active Duty in United States Army by Patients with Diseases of the Skin and Cellular Tissue (April 1, 1917, to December 31, 1919).*[2]

DISEASE	DAYS LOST
Carbuncle	43,000
Furuncle	212,800
Abscess	289,700
Cellulitis	223,200
Trichophytosis	45,300
Ectoparasitism	19,000
Dermatitis	12,600
Eczema	92,900
Erythema	20,500
Herpes	42,000
Impetigo	47,700
Lichen	2,200
Pityriasis	8,500
Psoriasis	42,700
Scabies	393,700
Other diseases of skin and cellular tissue	353,100
Total	1,848,900

It is not possible to discuss all the alterations in the cutaneous surface that may appear as a result of these diseases, but perhaps some service may be rendered by a review of some of the salient features of diseases with cutaneous manifestations that appear before medical officers under war conditions. For convenience, they have been divided into ten groups.

CUTANEOUS DISEASES CAUSED BY ANIMAL PARASITES

Parasitic diseases, both animal and vegetable, take the highest toll of disability from Army and Navy personnel. Other aspects of the parasitic diseases are often more serious than the infestation itself. Overtreatment, which has already been mentioned, constitutes a factor that definitely increases disability. The secondary pyogenic infections occurring as a complication of these parasitic dis-

eases at times assume serious aspects and are frequently a cause of prolonged disability. Perhaps more critical are diseases that the animal parasite may carry, so that the occurrence of such infestations is of particular interest from the standpoint of general preventive medicine.

Scabies ranks high in the list of these diseases that cause disability. When the disease is well marked, there is little difficulty in diagnosis. The increased itching at night, the characteristic locations of small papules and excoriations about the axillas, hands, wrists, genitals, waist and posterior upper thighs and the finding of burrows are sufficient evidence. The minimal infections in the early stages often offer considerable difficulty in the making of a definite diagnosis. In a case that has been overtreated, possibly with a mild secondary infection, a clear-cut diagnosis of scabies, pediculosis or a minimal urticaria is frequently difficult for an experienced dermatologist, and it is quite a problem for the average medical officer. Under military conditions, it is probably safer to treat such patients if there is sufficient suspicion of scabies; one should bear in mind, of course, the possibility that a dermatitis — a complicating and disabling factor — may result from therapy. Secondary pus infections of scabies are significant, and one should remember that the *Treponema pallidum* may enter through scabietic lesions.

Last year, several articles on scabies were written from the British standpoint. Buxton[5] brought out the fact that the interval from the egg to the adult female can be less than eight days and that the first egg may be laid twenty-four hours after maturity. The female is thought to lay approximately two eggs a day, perhaps a total of forty to fifty eggs. There may be an interval of eight to ten days between infestation and the development of symptoms. The disease usually comes from another human being, but animal infestations have occasionally been discovered, although it is not possible to distinguish morphologically between the human and animal parasites.

Carter[6] reviews the treatment of scabies with a sulfur Tather tablet, but only two groups of children were treated. MacKenzie[7] reviews the treatment of two groups of patients, one painted by a benzyl benzoate emulsion and the other by a sulfur ointment, indicating that the benzyl benzoate emulsion provided rapidity of cure, almost immediate relief from itching, ease and speed of application, and absence of unpleasant aftereffects.

Mellanby[8] experimented with the transmission of scabies to volunteers. Of 63 persons living in underclothes and sleeping in blankets used by known scabietic patients, only 2 became infected. On the other hand, a small number of experiments showed that scabies was conveyed by relatively slight personal contact. It is suggested that venereal contact provides better opportunity for the transmission of scabies than clothing.

Benzyl benzoate has had further favorable reports. Ingels[9] used a mixture of benzyl benzoate, sapo mollis and isopropyl alcohol in 90 patients, with good results, and Goldman[10] devised a cream modification that has proved effective, although it is more expensive than sulfur ointments.

In the Navy, Nolan[11] has used a soap paste, containing 18 per cent sulfur, that is efficient and inexpensive, but a three-day course of treatment is necessary. Rotenone has been reported to be successful in scabies, but Epstein[12] believes that it is ineffective in the presence of a complicating pyoderma.

Although these methods are successful in the treatment of scabies, they do not appear to be any more speedy and efficient than the present twenty-four-hour treatment with Danish ointment,[13] if the preparation can be compounded correctly and made available for use by the armed forces at the places where it is needed.

In any discussion of scabies, consideration must be given to the atypical cases, which may be difficult to detect, and to the minimal infestations, which are not sufficient to bring the patient to sick call. Search should always be made for contacts and sources of infestation, to eradicate them. Sterilization of clothing (bed and body) is essential. Too much emphasis cannot be placed on early detection and, perhaps, on the periodical inspection of troops for minimal lesions if a case or cases are discovered in a particular camp.

Pediculosis, of course, should be easy to recognize. The finding of nits in the scalp or the pubic region on careful inspection quickly confirms the diagnosis. The body cases, with minimal signs, are often difficult, and yet they may remain as a source of infestation to others. The secondary infections, as a result of infestation, will undoubtedly be markedly increased in the tropical cases, and antiseptic therapy, as well as antiparasitic therapy, should be undertaken. A 2.5 per cent solution of phenol applied for one hour and followed by combing with a fine-toothed comb is a satisfactory form of treatment for the scalp variety. The pubic cases clear quickly following shaving of the entire affected area. A shower and sterilization of clothing are necessary. Delousing stations, with bathing facilities and clothing sterilization, will adequately clear the body type.

General Diseases Caused by Animal Parasites

Emphasis should be placed on general diseases that are carried by animal parasites, especially those with cutaneous manifestations. Typhus[14] is a good example. With the acute onset and fever, there is a characteristic eruption of so-called "spotted fever," or petechial fever, which develops on the fourth or fifth day of the disease, new lesions appearing for forty-eight hours, when the eruption reaches its peak. The abdomen, chest, shoulders and back are attacked first, with a generalization of the eruption. There is usually a rose-red, macular eruption, varying in size from pinhead to bean, and these lesions frequently become hemorrhagic. The pure macular lesions disappear within a few days, and the others disappear in two or three weeks, depending on the amount of capillary transudation that takes place. This is frequently followed by scaling. In infested areas, the control of pediculosis requires vigorous attention by the medical officer in charge.

Rocky Mountain spotted fever[14] is another disease carried by an animal parasite, in this instance a tick. The eruption is usually more profuse, beginning as pinkish or reddish macular lesions, and appearing two to five days after the onset of the disease, usually on the arms and legs and later on the back. These lesions are apt to appear in crops. Often there are lesions in the mouth and throat, which may also become hemorrhagic and involve large areas. A considerable amount of desquamation may occur. Tick control will, of course, need to be emphasized in areas in which cases occur.

Tularemia,[14] appearing in the southern and central part of this country, shows characteristic, definite manifestations on the skin. The disease usually appears in persons who come in contact with wild rabbits, squirrels and so forth. It is quite possible that fleas, rodent lice and ticks may be carriers. A papule usually appears about three days after inoculation; it becomes painful, and nodular, gradually enlarges and eventually ulcerates, with fever, general lymphadenopathy and a febrile course. There are often severe constitutional symptoms, and another group of cases may present a rather generalized toxic eruption. The bacillus is often recovered, and stock strains of the bacillus are agglutinated by the patient's serum, usually at the beginning of the second week.

Bubonic plague, trench fever, relapsing fever and other diseases are also carried by animal parasites. Such bites on the skin, therefore, require far more consideration than the actual treatment of the individual soldier. These diseases require isolation, a search for other cases of the same disease, the destruction of the parasites, and a search for and control of the animal hosts—in other words, the initiation of a wide range of sanitary measures designed to carry out the accepted methods of preventive medicine for the particular disease.

Cutaneous Pyogenic Infections

The effects of the various pyogenic organisms on the cutaneous surface will occupy the attention, at some time or other, of every medical officer. One has only to note the figures for abscess, carbuncles, cellulitis, lymphangitis and so forth in the Surgeon General's report mentioned above for confirmation of this fact. Not only do these primary affections and impetigo, furunculosis, folliculitis, erysipelas and so forth, appear, but these conditions occur as complications of other skin disturbances and of overtreatment, and they may protract disability greatly. In the tropics, of course, heat and moisture raise the incidence of pyogenic infections very greatly. In Panama, last year, this point was emphasized to me by several of the medical officers with whom I talked.

Ammoniated mercury will undoubtedly be used widely for localized infections of the skin, and yet one must remember the possibility of sensitization to this drug and the large number of cases of dermatitis that have been associated with the indiscriminate use of the 5 and 10 per cent ointment.

The sulfonamide drugs have already altered the therapy of diseases in this group. Numerous articles[15, 16] have appeared in the literature, indicating the value of these drugs in the topical treatment of superficial pyogenic infections as well as that of chancroid, erysipeloid, lymphopathia venereum and so forth. Pillsbury et al.,[17] in a discussion of the bacterial flora of the skin, state that 5 per cent sulfathiazole in an oil-in-water emulsion or vanishing-cream base is more effective than in a grease base in combating various superficial coccal infections of the skin. Much more investigation needs to be done. Some skins have been irritated by these preparations; some persons have become sensitized to the drug. Numerous patients have shown increased sensitivity to sun after administration. There is not yet entire agreement among authorities regarding the best agent or the best vehicle for external application in these various infections, so that no general advice can be given on this subject. Caution must be used in the indiscriminate oral administration of this group for minor superficial infections.

To prevent pyogenic infections, emphasis should be placed on personal cleanliness for the soldier so far as it may be carried out at a particular time and place. Care should also be taken to detect minor pyogenic infections early, and to provide imme-

diate care for all wounds, great or small, so far as the urgency of the moment permits.

Cutaneous Diseases Caused by Vegetable Parasites

Mycotic infections will undoubtedly be prevalent. They will be found in any large group of persons, and in warm and moist countries, their incidence will rise very greatly. Involvement of the feet, hands, groins and other areas will occur. It should be borne in mind that all lesions in these areas are not necessarily mycotic, and laboratory confirmation of the diagnosis should be obtained if possible. Parasites of both the ringworm and the monilia groups are active in these areas, and may cause considerable disability. The 97,000 days lost by soldiers with trench feet in the last war[18] is an indication of the disability that can be produced, although perhaps a different kind of war will be waged this time. In civil life, many patients are seen in whom overtreatment and secondary pyogenic infections are important items in the disability. Every care should be taken to limit these complications in the Army and Navy.

It is well to remember that as yet no one therapeutic procedure will cure all cases.[19] Potassium permanganate soaks, wet dressings or Burow's solution often relieves the relatively acute and moist cases, and some modification of Whitfield's ointment is very satisfactory in the more chronic and scaling cases. Vaccines have not proved effective.[20] It is essential to give due consideration to the type and stage of the disease, the type of skin involved and the reactions to previous therapy. It is desirable for the patients with the severer cases to be hospitalized early, under the charge of a dermatologist, if possible.

Sulzberger, Baer and Hecht[21] have recently expressed an opinion that exposure to patients with active phases of ringworm disease has played little part in producing attacks. They believe that patients may carry pathogenic fungi in quiescent foci on feet, toenails and elsewhere, and that they have exacerbations when certain contributory factors lower their resistance to their own germs. In other words, a variation in the degree of host resistance is the determining factor in the production of clinical activity. The authors found, for example, that there were very few cases of proved conjugal or familial transmission of ringworm of the feet and groins. They emphasize the need for personal care and hygiene of the feet and groins as having the best prospect of success in preventing attacks, and they minimize the present, widely used methods of prophylaxis as not only illogical but also potentially harmful. Here again, mention is made of the possibility of secondary infection and of the extreme harm that may be done by overtreatment of the skin.

Exanthems

It should not be forgotten that the manifestations of the exanthems may present themselves for interpretation at any sick call. The typical case is easily recognized, but the atypical case, the abortive case and the case with the incomplete quota of typical symptoms may well puzzle the best of diagnosticians. The cutaneous manifestations of scarlet fever may well be simulated by the eruptions of toxic erythemas, such as those caused by food, focal infections and drugs. Quinine, belladonna and salicylates are especially likely to produce scarlatiniform eruptions, although the rashes produced by a given drug vary widely in different persons. Even the eruption after arsphenamine, especially that occasionally appearing about the ninth day after injection, may simulate a scarlet-fever eruption. Measles is quite characteristic as a rule, but the eruptions of the barbiturates and the sulfonamides are often morbilliform. Syphilis or pityriasis rosea in the early stages, if profuse and of sudden onset, may present an appearance on the skin similar to that of measles. Varicella should rarely need to be differentiated from variola in a completely vaccinated army. The principal difficulty will arise in the recognition of the mild and abortive case with comparatively few lesions. German measles less often presents problems in diagnosis, because of the acute onset and often generalized involvement, with few other symptoms. Epidemics of this disease may be a definite source of time lost from military duty.

In any event, with any suspicious case, isolation until a definite diagnosis can be made is the best rule to follow.

Virus Infections

Among the virus infections that may be seen are innumerable varieties of warts or verrucae, for which destructive measures of one kind or another will be necessary.

Herpes zoster and herpes simplex will also be seen. No specific therapy has yet been developed, although some cases of herpes zoster have done extremely well and their courses have been shortened by autohemotherapy, as well as by daily injections of small doses of pituitary extract. The use of vitamin B_1 has not been generally successful. The possibility that herpes zoster may appear during or after a varicella epidemic, or that varicella-like lesions may appear in herpes zoster, should be remembered.

Tropical Skin Diseases

Tropical skin diseases will need consideration by American physicians more than in any other war. Many varieties of cutaneous disease are due to fungi, protozoa and metazoa. The manifestations vary with the particular section of the world to which a medical officer may be assigned, and no definite review of these diseases is possible. Strong's[14] recent revision of Stitt's well-known book is worth studying for those ordered to tropical countries. The secondary pyogenic infections of these infestations will undoubtedly be a further cause of disability. Adequate precautions will need to be taken to prevent the invasion of this country by these diseases as the troops return home.

Disturbances Caused by Vesicant Chemical-Warfare Agents

Chemical-warfare agents caused considerable injury and some deaths during the last war. The principal vesicants are mustard gas and lewisite. Mustard gas, recognized clinically by the garlic-like odor and brownish oily stains on the clothing, is of low volatility, and may remain in the open for ten days — longer in the woods and in cold weather. After a latent period of two to six hours, the gas causes erythema with itching and burning, and the absorption of the liquid begins in three minutes if it contacts the skin. Vesicles and bullae are rapidly produced and heal slowly. In contrast to mustard gas, lewisite, which has a geraniumlike odor, has a shorter latent period, — fifteen to thirty minutes, — and there may be more sneezing and nasal irritation. The bullae form more quickly, and because of the arsenic content, varying degrees of arsenic poisoning are produced by the cutaneous lesions. With both, if the patient is seen early, the removal of clothing for decontamination is necessary, and the skin should be treated with solvents (kerosene, gasoline and so forth) or oxidizing agents (bleaching-powder paste, dichloramine T, hydrogen peroxide and sodium bicarbonate).

Goldman and Cullen[22] have recently reviewed the effects of these agents and their treatment. The subject is also presented in a seventy-five-page booklet issued by the Office of Civilian Defense.[23] The later treatment of burns by these agents is well outlined by MacCollum.[24] If assigned to front-line duty, the medical officer should be familiar with the protective methods against these agents, their first-aid care, their aftercare and the details of decontamination.

Syphilis

Syphilis deserves further comment. Early primary and secondary syphilis will undoubtedly be seen. It should be detected early and treated adequately. The cases should be sent to special centers for treatment until noncontagious, and provision should be made for uninterrupted continuous treatment[25] in accordance with accepted procedure. If the war lasts five years, attempts to conserve manpower by adequate early treatment are particularly necessary. At present, the new five-day treatment[26] or one of its modifications is not for routine use. A clinic might be set up, however, in some large center, where, under the direction of physicians experienced in the proper technic, selected cases could be given the advantage of this method. The part that the medical officer can play in the control of venereal disease is valuable from the standpoint of preventive medicine. Early detection and diagnosis, tracing and elimination of the source of the infection, and adequate treatment and follow-up are the keystones in the prevention of disability due to syphilis and other venereal diseases.

Chancroids may be confused with the primary lesion of syphilis. Dark-field examinations, until the diagnosis is clear, and serologic follow-up should be done to prove that syphilis was not acquired at the same time. Evidence is increasing that sulfanilamide is beneficial in many cases of chancroidal infection.[27, 28]

In the differentiation of penile lesions, one should remember the value of skin tests — the Ito-Reinstierna test for chancroid[29, 30] and the Frei test for lymphopathia venereum. In the treatment of the latter disease, sulfanilamide has proved of value.[31]

Other Skin Diseases of Young Men

Numerous cases of other relatively common diseases will occur in the young men inducted into the Army or Navy as essentially healthy persons. There will be many cases of dermatitis from the many contacts made in the field or in camp. A dermatitis may appear anywhere, it may be obstinate and may cause much disability, and much detective work, to ferret out the exact causal factor, may be required. It is essential that soothing treatment be given at the beginning, or when the diagnosis is in doubt. Light[32] has well outlined the therapy of acute undiagnosed manifestations on the skin.

Overtreatment was mentioned above, but it deserves emphasis again. Dermatitis from therapy, whether it is due to self-medication without a proper diagnosis, to improper medication from an error in diagnosis, to the excessive application of an appropriate remedy or to hypersensitivity of the patient, causes more visits to the dermatologist than any other single skin disease. Miller, Ayres and Alderson[33] point out that many patients seek relief because of such treatment and not because of their primary complaint. A longer period of disability

is produced because the dermatitis must be quieted down before the original disease can be properly treated.

Atopic eczema — with the involvement particularly of the flexures, sides of neck and face, and history of hay fever, asthma, previous attacks and allergic family history — will undoubtedly be seen in the colder portions of the world and under stress and strain. Most of these patients, especially if there is much involvement, ought to be returned to civil life, because there can be much disability with this disease; they should not be inducted into military service.

Acne will, of course, appear, but it is not necessary to review the various forms of therapy that may be required in the treatment of these cases.

Psoriasis may develop further in persons who had a minimal amount on induction, and if severe types appear, it is probably best that the patients be discharged from service, although it is possible that service in the tropics, with sun, may be of aid in many cases. Exacerbations of this disease should probably be treated in a general hospital rather than in the field or camp.

Sycosis vulgaris, a pyogenic folliculitis of hairy areas, is a pyogenic disease that appears in young men. The characteristic follicular pustule is usually evident. The acute type usually responds quite well to antiseptic therapy, but recurrence of attacks should probably call for discharge from the Army, since there is a possibility of much disability.

Other diseases will be seen by the medical officer, but these are the commonest conditions that have not already been mentioned.

* * *

Much disability in the armed forces of this country may result from dermatologic diseases if previous experience is any criterion. A considerable reduction in the days lost from these causes may be attained if consideration is given to the following general suggestions:

Encourage early report at sick call for skin disturbances.

Pay careful attention to minor trauma and minor skin ailments.

Remember the possible seriousness of parasitic infestations.

Watch for the onset of secondary pyogenic infections.

With an unusual picture of the skin, review the possibility of exanthems, drug eruptions, syphilis and the less frequent self-induced eruptions.

If a consultant is available, use him frequently.

Apply soothing treatment for acute conditions and for dermatoses in which a diagnosis is uncertain.

Avoid overtreatment.

If a patient does not respond to treatment quickly or if the condition becomes worse, refer him, if possible, to a general hospital early to avoid long disability.

To salvage the soldier with acquired syphilis, diagnose the condition early and provide adequate treatment for at least one and a half to two years.

Remember that full participation in the venereal-disease control program is the responsibility of every medical officer with troops.

The prevention of disability is paramount.

References

1. *The Medical Department of the United States Army in the World War.* Vol. 9. *Communicable and Other Diseases.* 628 pp. Washington: Government Printing Office, 1928. P. 551.
2. *The Medical Department of the United States Army in the World War.* Vol. 15. Part 2. *Medical and Casualty Statistics.* 1368 pp. Washington: Government Printing Office, 1925. P. 226.
3. *The Medical Department of the United States Army in the World War.* Pp. 292-294.
4. Sulzberger, M. B., MacClatchie, L. K., and Parsons, R. P. Military dermatology and syphilology. *M. Clin. North America* 11:1677-1698, 1941.
5. Buxton, P. A. Parasitology of scabies. *Brit. M. J.* 2:397-401, 1941.
6. Carter, D. L. Treatment of scabies: use of sulphur lather tablets. *Brit. M. J.* 2:401-403, 1941.
7. MacKenzie, I. F. Scabies treated by a benzyl-benzoate emulsion. *Brit. M. J.* 2:403-405, 1941.
8. Mellanby, K. Transmission of scabies. *Brit. M. J.* 2:405, 1941.
9. Ingels, A. E. Scabies: its treatment with benzoyl benzoate, as compared with sodium thiosulphate plus hydrochloric acid. *California & West. Med.* 50:265-267, 1939.
10. Goldman, L. Cream modification of the benzyl benzoate treatment of scabies. *Arch. Dermat. & Syph.* 39:878, 1939.
11. Nolan, R. A. Sulfur soap paste in the treatment of scabies. *Arch. Dermat. & Syph.* 36:846, 1937.
12. Epstein, E. Treatment of scabies with rotenone. *Arch. Dermat. & Syph.* 45:950-952, 1942.
13. Greenwood, A. M., and Reilly, M. Treatment of scabies with the so-called Danish method. *Arch. Dermat. & Syph.* 35:602-605, 1937.
14. Strong, R. P., *Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases.* 2 vol. 1747 pp. Philadelphia: P. Blakiston's Son & Co., 1942.
15. Beinhauer, L. G., Knoll, A. F., and Perrin, S. R. Clinical response of certain dermatologic diseases to sulfathiazole. *Arch. Dermat. & Syph.* 43:621-627, 1941.
16. Combes, F. C., and Canizares, I. Sulfanilamide and allied compounds: their value and limitations in dermatology. *Arch. Dermat. & Syph.* 44:236-247, 1941.
17. Pillsbury, D. M., Livingood, C. S., and Nichols, A. C. Bacterial flora of the normal skin: report on the effect of various ointments and solutions, with comments on the clinical significance of this study. *Arch. Dermat. & Syph.* 45:61-80, 1942.
18. *The Medical Department of the United States Army in the World War.* P. 214.
19. Symposium on the practical management of eczematous ringworm of the hands and feet ("athlete's foot" — dermatophytosis and dermatophytids). *J. Invest. Dermat.* 3:523-562, 1940.
20. Tolmach, J. A., and Traub, E. F. Treatment of dermatophytosis with vaccines. *Arch. Dermat. & Syph.* 38:925-929, 1938.
21. Sulzberger, M. B., Baer, R. L., and Hecht, R. Common fungous infections of the feet and groins: negligible role of exposure in causing attacks. *Arch. Dermat. & Syph.* 45:670-675, 1942.
22. Goldman, L., and Cullen, G. E. The vesicant chemical warfare agents. *Arch. Dermat. & Syph.* 42:123-136, 1940.
23. *Protection Against Gas.* 75 pp. Washington: Office of Civilian Defense.
24. MacCollum, D. W. The early treatment of burns. *New Eng. J. Med.* In press.
25. Moore, J. E., Kemp, J. E., et al. *The Modern Treatment of Syphilis.* Second edition. 674 pp. Springfield: Charles C Thomas, 1941.
26. Baehr, G., et al. Symposium on massive arsenotherapy in early syphilis by the continuous intravenous drip method. *Arch. Dermat. & Syph.* 42:239-284, 1940.
27. Canizares, O., and Cohen, I. A. Treatment of chancroid with sulfanilamide. *Arch. Dermat. & Syph.* 42:649-652, 1940.
28. Culp, O. S. Treatment of chancroid with sulfanilamide. *Am. J. Syph., Gonor. & Ven. Dis.* 24:622-631, 1940.
29. Robinson, H. M. Analysis of intracutaneous tests on 309 patients for lymphopathia venerea and chancroid. *South. M. J.* 33:144-154, 1940.
30. Brandt, R., and Tarpin, R. Dependability of skin tests in the diagnosis of lymphopathia venerea and chancroid, especially in the colored race. *Am. J. Syph., Gonor. & Ven. Dis.* 24:632-642, 1940.
31. Costello, M. J., and Cohen, I. A. Lymphogranuloma venereum observations in 187 cases at Bellevue Hospital. *Arch. Dermat. & Syph.* 44:391-397, 1941.
32. Light, S. E. Palliative treatment of acute undiagnosed skin diseases. *Northwest Med.* 40:92, 1941.
33. Miller, H. E., Ayres, S., Jr., and Alderson, H. E. Overtreatment in dermatology. *California & West. Med.* 51:251-253, 1939.

NEW HAMPSHIRE MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND FIFTY-FIRST ANNUAL MEETING

House of Delegates, May 11, 12 and 13

(Concluded)

After commending the committee for its excellent work, Dr. Sycamore, of the Committee on Officers' Reports, recommended that the House of Delegates express its sincere appreciation of the work of the Committee on Maternity and Infancy, and extend its congratulations to the members of this committee for their high standard of achievement; it was voted to adopt this portion of the report.

At this point, the reading of the reports was interrupted to permit a discussion by Dr. Burroughs, who spoke on the New England Institute. This is strictly an educational institute and is intended to provide some theoretical training in public health to local health officers, to the workers of various local volunteer agencies, to the staff of the state departments of health and to any others who may be interested. It is particularly valuable because many of these people have no opportunity for formal training in public health.

In the ordinary rotation, next year will be the year for the institute to come to New Hampshire. It was held in New Hampshire in 1926. From 1930–1938, it was dropped, but it was revived in 1939 in one section, and in 1940, it was fully revived, and has been held every year since then. The 1942 session has just been held in Providence, Rhode Island, with an attendance of 972 from the six states of New England.

The State Board of Health voted to recommend to the Governor and Council that this institute be held in New Hampshire in 1943; that recommendation was laid before the Governor and the Council for consideration.

At the meeting held at Hartford, Connecticut, in 1940, the Connecticut State Medical Society was one of the co-sponsors, and was responsible for one of the sectional meetings, which occupied the afternoon of one day of the institute.

Dr. Burroughs asked if, in the event that this meeting should be held in New Hampshire next year, the New Hampshire Medical Society would be interested in co-sponsoring and in putting on a sectional meeting. No expense would be entailed by the Society, because the State Board of Health would handle the cost, as well as the details, on behalf of the Secretary of the New Hampshire Medical Society.

President Dolloff asked what being a sponsor entailed.

Dr. Burroughs replied that it constituted having the name printed on the programs and officially endorsing the institute.

Dr. D. G. Smith moved that the Society be a co-sponsor of the New England Health Institute, the details of the participation of the Society to be left to the Committee on Public Health.

Dr. Metcalf stated that Dr. Burroughs had told him that the State Board of Health would be very glad to arrange the program. However, Dr. Metcalf did not know the health officials who ought to be on the program. There are three days, and the sponsor is responsible for half a day. The Board of Health, in co-operation with the Committee on Public Health, could pick out the men who are going to speak and place them on the program.

The motion presented by Dr. Smith was then seconded and carried.

Dr. Parsons asked if any delegate knew of any members of the county societies who would be interested in taking a course covering the medical aspects of chemical warfare, at either the University of Cincinnati or another teaching center.

The report of the Committee on Medical Economics was then presented.

Report of the Committee on Medical Economics

The chairman of the Committee on Medical Economics, Dr. Clarence E. Dunbar, has been called into active service with the Army Medical Corps, and the work has been carried on since his departure by the remaining two members. We have greatly missed Dr. Dunbar's wise counsel in our deliberations.

The committee has directed its attention to three major problems the first of which is a medical-service corporation.

In pursuance of the instructions received from the House of Delegates, your committee has conferred with Attorney Sulloway regarding an enabling act to permit the establishment by the New Hampshire Medical Society of a Medical-Service Corporation in New Hampshire. Attorney Sulloway has drawn up a proposed act, with due reference to the recommendations of your committee and the laws and regulations governing the operation of the insurance corporations in New Hampshire, and a copy is appended to this report. Your committee has scrutinized this act and believes that it embodies the provisions that have been

proved by experience in other states to be essential to the successful working of such a plan. We wish to commend the careful and conscientious effort that Attorney Sulloway has given to this task.

Your committee recommends that the New Hampshire Medical Society approve the proposed Act Governing Medical-Service Corporations and instruct the Committee on Public Relations to secure its introduction into the next session of the Legislature.

It is to be emphasized that the passage of the act by the Legislature would in no way obligate the Society to embark on a program of prepayment medical care, but would merely establish the legal setup under which such a program could be instituted at any time in the future when such a step should seem advisable.

The work of the National Physicians' Committee for the Extension of Medical Service is of continuing importance and will be of greater value as time goes on. The trend toward governmental regulations of medical practice was gaining rapid momentum until interrupted by the war emergency. No thoughtful observer doubts that this trend will be resumed at the conclusion of the war; it will be accelerated, in fact, by the economic problems of the postwar period. The preservation of the personal relation between physician and patient, which we believe to be the most efficient and effective form of medical care, depends largely on the efforts of the medical profession to educate the public to awareness of its value. The National Physicians' Committee has proved itself an effective instrument for this purpose, and as such deserves the support of the medical profession.

The budget of the National Physicians' Committee would be met by a contribution of one dollar from each member of the American Medical Association, and this seems a small enough amount for each of us to invest in his own future. Your committee recommends that the House of Delegates request each component county society to make such a contribution to the treasurer of the New Hampshire Medical Society, to be forwarded to the National Physicians' Committee as a contribution from the physicians of New Hampshire; and that a committee of the Society be constituted to co-operate with the National Physicians' Committee in its educational work and in the securing of the necessary contributions for its support. The Committee on Public Relations might logically be designated to carry out this task.

Your committee has wrestled with the problem of compensation for the physician in service, but it believes that the more it wrestles, the more it becomes enmeshed in the coils of this knotty question. It is certain that no recommendation can be formulated that will be universally applicable to every situation. In some circumstances, it would be only just that part of the fees collected from the patients of a colleague who is in the service should be remitted to him. Such action must be left, however, to the judgment and conscience of each physician. Consideration is being given to the possibility of issuing a limited license to any physician coming into the State to take over the practice of one who has entered the service — such a license would be valid only for the duration of the emergency. Your committee has no specific recommendations to make, however, regarding this complex problem.

The Committee on Medical Economics was instructed at the last meeting of the House of Delegates to investigate the problem of medical care for low-income families in rural communities. This problem, however, has apparently been solved by the changing economic situation, and the State Grange believes that there is no need for such an investigation at the present time.

LESLIE K. SYCAMORE, *Acting Chairman*
RICHARD W. ROBINSON

Dr. Sycamore moved that the Committee on Public Relations be instructed to secure the introduction of the enabling act into the next session of the Legislature.

This motion was duly seconded and was carried.

Dr. Sycamore then moved the adoption of the portion of the report relating to the work of the National Physicians' Committee.

Dr. Metcalf asked how this money would be turned over to the National Physicians' Committee.

Dr. Sycamore replied that the treasurers of the county societies would turn it over to the treasurer of the New Hampshire Medical Society.

The motion presented by Dr. Sycamore was duly seconded and carried.

Dr. Sycamore then moved that the House of Delegates approve the principle of limited licensure of physicians who move to the State to replace those who enter the armed forces.

This motion was duly seconded and carried.

President Dolloff presented the report of the Committee on Mental and Social Hygiene.

Report of the Committee on Mental and Social Hygiene

There have been no new developments in New Hampshire in the field of mental and social hygiene during the past year, nor are there likely to be for some time to come. Specialists in the field of mental hygiene are making what contribution they can to the effort to prevent another billion-dollar expense to the Government, such as that resulting from the previous war because of the comparatively large number of prepsychotics and other misfits taken into the Army.

The institutions expect to take a severe setback in both their intramural and extramural activities. With the competition offered by the Federal government in munitions plants and other employing agencies, the institutions can hardly be expected to retain or replace good, intelligent employees who can easily obtain work at more pay and fewer hours elsewhere.

Under ordinary circumstances, we should encourage early commitments, but under present conditions we suggest that no one be sent to the institutions who can be cared for elsewhere.

Since the microcephalic patients usually have ossified cranial sutures at birth, the officials at the State School are convinced that medical schools should prepare physicians to recognize the grosser degrees of mental defect at birth, and that they should be reported to the Board of Health as such. Although notification is made of a case of measles, which lasts ten days, no record is made of the birth of an idiot, who is a living first mortgage on the State for life.

This state has an excellent sterilization law, and if a sane approach to the subject is maintained, it should re-

main a very useful aid in our battle with mental deficiency and mental disease.

Since there is no particular connection between mental and social hygiene, and since each calls for a different line of thought and investigation, we recommend that a separate committee for social hygiene be created by the House of Delegates.

The New Hampshire Hospital Association has a committee on legislation, whose function it is to keep track of proposed legislation affecting hospitals, but there does not appear to be any similar committee of this Society.

CHARLES H. DOLLOFF, *Chairman*
BENJAMIN W. BAKER
JOHN B. MCKENNA

Dr. Sycamore stated that the Committee on Officers' Reports agreed that social hygiene is a separate field from mental hygiene, and moved that questions of social hygiene be placed under the jurisdiction of the Committee on Public Health, and that, at the next revision of the by-laws, the words "and Social" be struck from the title of this committee.

Dr. Dye asked whether the revision of the by-laws should not be referred to the Committee on Constitution and By-Laws.

Dr. Sycamore answered in the affirmative, and amended his motion accordingly.

The motion was then duly seconded and carried.

Report of the Committee on Tuberculosis

It is with deep regret that the Committee on Tuberculosis records in this report the death on January 29, 1942, of one of its members, Dr. Robert M. Deming, late superintendent of the New Hampshire State Sanatorium at Glencliff. Dr. Deming had been a member of this committee for many years. He had served the State of New Hampshire as superintendent of the sanatorium at Glencliff from 1922 until his death.

For twenty years, Dr. Deming had given unstintedly of himself in devoted service to his patients. Under his leadership and administration, the sanatorium had been developed into one of the Nation's best for the treatment and cure of tuberculous patients. He was a valiant worker in the campaign for the early detection, prevention and treatment of the disease. His death is a serious loss to the tuberculosis campaign in our state.

It is interesting that within the past few years an active campaign has been under way for the organization of tuberculosis committees in state and county medical societies throughout the Nation. Only recently, the Pennsylvania State Medical Society announced the organization of a state tuberculosis committee in its membership.

It is probably true that the New Hampshire Medical Society can claim the distinction of having organized the first tuberculosis committee in a state medical society. This committee was appointed in 1914.

Throughout the years, your committee has worked with the following objectives in view: to stimulate active interest in combating tuberculosis on the part of physicians in general practice; to establish a harmonious and helpful relation between the medical profession and official and voluntary organizations engaged in the control of tuberculosis; and to keep the profession informed concerning resources in the State for the diagnosis, prevention and treatment of the disease and of progress toward its control and eradication.

The encouraging progress that has been made in combating tuberculosis in New Hampshire is due in large measure to the active support and aid of the members of the New Hampshire Medical Society.

In 1940, the tuberculosis mortality in the State fell to a new low figure of 21.2 per 100,000 population. In 1916, the death rate was 114.5. Only six states in the Nation had a lower death rate than New Hampshire — Utah, Wyoming, Iowa, Nebraska, Idaho and North Dakota. According to a statistical study made by the National Tuberculosis Association of the percentage of reduction in tuberculosis death rate among the states of the union in the period 1916 to 1940, New Hampshire presented the greatest reduction — 81.5 per cent. This splendid result is due to the united effort of our physicians, sanatoriums, health and school departments, tuberculosis agencies and the people throughout the State.

As always, our problem is to find tuberculosis cases in all stages of the disease, best of all in the earliest stages and even before activity is present.

An excellent piece of tuberculosis case finding is being carried on at the Selective Service Induction Center in Manchester. An essential part of the examinations at this induction center is an x-ray examination of the chest for every man. We are not permitted to give out figures regarding the number of men called, or other statistics, but it can be stated that because of these chest x-ray examinations, tuberculosis is being kept out of our military forces and that the entire program is aiding materially in the discovery of tuberculosis in the State. All men rejected because of tuberculosis are reported to the State Board of Health, and follow-up service is provided by the New Hampshire Tuberculosis Association.

For some years, your committee has emphasized the great value of the chest x-ray film in the discovery of tuberculosis. It is known that the lesions can be seen on the chest x-ray film before signs can be heard by the stethoscope. We again urge on the members of the New Hampshire Medical Society frequent use of the chest x-ray examination. The New Hampshire Tuberculosis Association is ready to provide chest x-ray films for persons referred by physicians when these persons are unable to pay for them.

We urge prompt examination of all members of the family when a case of tuberculosis is found, since in a household where an open case has been discovered, we usually find that all members are positive tuberculin reactors.

We urge at least a tuberculin test for all persons caring for children. This should be a routine procedure for nursemaids, governesses and others in contact with children.

Bed rest continues to be emphasized in treatment. In most sanatoriums, on admission of patients, all are on bed-rest regime for study by the physicians and decision about activity of the disease.

In addition to rest treatment, various forms of chest surgery, such as pneumothorax, phrenicectomy, phrenicotomy and thoracoplasty, continue to be emphasized as aids to cure of the disease.

In this connection, it seems advisable for the best interests of the chest surgical services in our two sanatoriums that this program of treatment be developed under the direct charge and supervision of one chest surgical service.

By means of this procedure, possible chest surgical cases would be reviewed oftener and followed up more thoroughly so far as the carrying out of recommendations for treatment is concerned. Also, the responsibility would be more definitely allocated. Furthermore, in conjunction with this plan, a definite program of providing costs of treatment could be developed.

One of the members of the committee made a study this spring in Michigan of the chest surgical services in that state. The program is carried out along the lines we have suggested as being advisable in New Hampshire. In Michigan, the program has been thoroughly developed, is now in operation for some years, and appears to be working successfully.

For some time, it has appeared that the policy of restricting admissions to one of the two sanatoriums in the State to far-advanced pulmonary tuberculosis cases has not been satisfactory. Theoretically, a sound policy, in practice it has resulted in lowered morale in the institution and reluctance of patients to enter it. During the past three years, in this one sanatorium, 75 deaths occurred — almost half within sixty days after admission.

During the past year, the New Hampshire Tuberculosis Association has been making a study of this problem. In the summer of 1941, the association sent a questionnaire to the executive secretaries of the state tuberculosis associations of the United States asking information regarding presence of a similar problem in their respective states and how it had been solved. Forty-four states sent replies. Some few were working on the problem: all the others had had similar experiences and had solved it by dividing their states into districts or sections and by admitting patients in all stages of the disease to the sanatorium in the district or section in which they live. It is our opinion that this policy should be carried out in New Hampshire.

The New Hampshire State Sanatorium and the New Hampshire Tuberculosis Association continue to offer their services in the interpretation of chest x-ray films sent to them for diagnosis.

The Nation is engaged in a great world war. Today, tuberculosis is more than a threatening shadow. It is a definite obstruction to victory, acting like sand in the gears of the production and fighting machine. Longer hours of labor and increased mental and physical strain tend to increase tuberculosis. The infection is still widespread among our people. Tired bodies may permit ever-lurking germs to attack the lungs successfully.

There must be no letup in the campaign against the disease. In fact, the fight must be carried on with ever greater aggressiveness to hold the gains won.

Your committee wishes to express its appreciation for the wholehearted assistance and encouragement accorded to it by the members of the New Hampshire Medical Society.

ROBERT B. KERR, *Chairman*
M. DAWSON TYSON

Dr. Sycamore, after commending the committee for its work, moved that the House of Delegates approve the recommendations of the Committee.

This motion was duly seconded and was carried.

Dr. Robertson read a letter from the American Medical Association Bureau of Medical Economics, addressed to the secretary of the Society, regarding H. R. 4476, now in the Senate Committee on Military Affairs. He moved that the recommendation contained in the letter be seconded by the Society, that such notices be sent to the Committee on Military Affairs, and that a personal note be sent to Senator Bridges, who is a member of the Committee on Military Affairs.

This motion was duly seconded and carried.

Dr. Robertson then read a letter concerning the proposed tax on hospitals and colleges, which may be enacted. The tax referred to would affect the incomes of educational and charitable institutions derived from funds received from real-estate investments. Dr. Robertson stated that the Committee on Communications and Memorials recommended that the secretary of the Society be instructed to notify the congressmen and senators that the Society, as a body, protested any abridgement of the exemptions now allowed by Section 101 of the revenue laws regarding charitable and educational institutions.

This motion was duly seconded and carried.

Dr. D. G. Smith then made his report as delegate to the American Medical Association.

Report of the Delegate to the American Medical Association

The ninety-second annual session of the American Medical Association was held at Cleveland, June 2–6, 1941. It was a well-attended and interesting meeting, at which your delegate served as a member of the Committee on Executive Session. The Distinguished Service Award was given to James Ewing of New York; and Fred W. Rankin of Lexington, Kentucky, was chosen president-elect. There was considerable discussion of specialty boards for the general practitioners, and there was also a demand for a new section of the scientific assembly for this group. It was decided to have an experimental "session" for general practitioners in the Section on Miscellaneous Topics at the next session. This is to be held at Atlantic City, June 8–12, 1942, and is to be known as the Pan-American Session.

A report was made of the indictment and trial of the American Medical Association, and it was voted by the House of Delegates that the Trustees be instructed to direct counsel to appeal the judgment based on the verdict of guilty against the Association.

Medical preparedness was the chief topic of discussion at the meeting. A resolution requesting the Surgeons General of the Army and Navy to make women physicians eligible for reserve commissions was disapproved, although the spirit of American medical women in offering their services to the Government was applauded. Payment for services of the physicians serving with the Selective Service System was disapproved. A resolution was adopted requesting the establishment by the federal government of a Procurement and Assignment Agency. It was proposed that this agency should continue the work of the Medical Preparedness Committee of the American Medical Association, and that it should have authority for the selection of physicians who would be necessary for immediate duty and who would be called from civilian practice to serve with the armed forces. Such an agency has been established, is known as the Procurement and Assignment Service, and is in operation at the present time. Its function is to procure physicians for the

armed forces, and at the same time to be certain that the medical needs of the civilian population are taken care of.

DEERING G. SMITH

Dr. D. G. Smith then presented the report of the Committee on Medical Preparedness.

Committee on Medical Preparedness

The Committee on Medical Preparedness has continued its co-operation with the Selective Service System and has approved all appointments of physicians as examining physicians and members of the medical advisory boards. Most of the work of this committee has been taken over by the Procurement and Assignment Service, with Dr. Deering G. Smith as state chairman; Drs. Ezra A. Jones and John P. Bowler serve with him. This service has the twofold purpose of securing physicians for the armed forces and providing for the care of the civilian population. The availability for active service of each physician in the State is to be determined. No federal appropriation of money has been made to carry on this work, although it is hoped that such an appropriation will be made in the near future. Some state medical societies are going to continue to finance the project, whereas others are planning to accept federal funds. Your committee desires the decision of the House of Delegates.

An intensive recruiting drive is about to be started in this state, to secure fifty physicians for the army before July 1, 1942. Captain E. L. Wiemers, representing the Office of the Surgeon General, and Lieutenant R. G. Vedeler, representing the Office of the Adjutant General, will be working with Dr. Smith, and it is expected that commissions will be granted within a week of the time of application.

DEERING G. SMITH, *Chairman*
EZRA A. JONES
CARLETON R. METCALF

Dr. Sycamore, acting for the Committee on Officers' Reports, recommended that the House of Delegates express to Dr. Deering G. Smith its sincere appreciation of the whole-hearted and unselfish enthusiasm with which he had devoted himself to the exacting and often thankless duties of the Procurement and Assignment Service, giving freely of his time and energy, although frequently at no inconsiderable inconvenience to himself and detriment to his own practice.

For the work of the Procurement and Assignment Service, a large number of detailed records must be kept; a great deal of correspondence is entailed, and the frequent use of the telephone and telegraph is necessary. Obviously, no budgeting of such items is possible. Dr. Sycamore therefore recommended that the Treasurer be instructed to defray all necessary expenses incident to the work of the committee, and that reimbursement be made for expenses already incurred.

After much discussion, a motion by Dr. Robertson — that the Government be allowed to pay the expenses of the Procurement and Assignment Service — was lost. Dr. Sycamore then moved that the expenses incurred by Dr. Smith be paid out of the general funds of the Society.

This motion was duly seconded by several of the members present and was carried.

Dr. Gray then spoke on the status of chiropractors in New Hampshire. He stated that at the spring meeting of the Rockingham County Medical Society, the delegates were instructed to bring before the meeting a recommendation, which was as follows:

> The Rockingham County Medical Society is unalterably opposed to the practice of chiropractic. The kindest thing one can say for it is that it is quackery.
>
> It is obvious that only by pressure politics could chiropractic have obtained a foothold in New Hampshire. It can be ousted only by pressure politics. This will mean that the Society is willing to expend money and employ legal talent to conduct a successful fight.
>
> The delegates of Rockingham County are instructed to bring this matter before the New Hampshire Medical Society. As a Society, we favor a study of this problem, with a view to definite action in the coming legislature. We favor any legitimate action, even at considerable expense, that can bring about a revocation of chiropractic in New Hampshire.

Dr. Metcalf stated that the chiropractors usually have two or three members in the Legislature, and that the recommendation proposed would probably only make enemies and would not succeed in forcing the chiropractors out of business.

Dr. H. O. Smith then stated that the detailed report of the Trustees would be given on Wednesday afternoon, but that some financial matters should be touched on at this meeting, which was probably the most largely attended meeting of the House of Delegates. He then made the following remarks:

> As most of you are aware, all the invested funds of our Society have, until last July, been deposited in various savings banks. A year ago, the Board of Trustees thought it wise to diversify our investments further, the determining factor being the strong effort then being made by the national authorities to sell savings bonds of various types.
>
> Our purchases were of Series G bonds, a registered current income security paying 2½ per cent a year in semiannual installments, the funds being taken from accounts and banks that seemed to us advisable.
>
> You should know that this transfer was made without the loss of interest for a single day, and that these bonds are yielding a slightly higher rate of interest than was being paid by the banks. But, in view of present conditions, the important thing is that the New Hampshire Medical Society now has $10,000 invested in the future of America.

Dr. Sanders then spoke as follows:

> Last year, I had the honor of being elected vice-president at the meeting of the House of Delegates. Because a number of our physicians will enter the armed services, I believe that I shall be working to the limit of my strength next year; therefore, and because the last two winters have been rather trying to me, I shall not be able to assume the burden of the duties

of president of the New Hampshire Medical Society, and I do not wish to be a candidate for the office of president. This year will be a legislative year, and it is sometimes hard to get from Claremont to Concord. I believe that I should not take this office.

Dr. Wilkins expressed his regret that Dr. Sanders declined the office.

Dr. Gray stated that an important matter brought up by the Rockingham County Medical Society was the practice by naval doctors, particularly in Portsmouth. Those men stationed at the Navy Yard are engaging in civilian practice in their hours off, and they wanted to know what their status was. Dr. Gray had written a letter to the Board of Registration to see whether or not a license was required. In the Army, medical men have definite instructions not to indulge in civilian practice unless they are stationed in their own communities. That apparently was not so in the Navy, because some of the men in Portsmouth are indulging in practicing medicine; the members of the Rockingham County Medical Society wanted to know if the House of Delegates could take some action or make some recommendation.

Dr. Burroughs stated that he had discussed the matter with the naval authorities about three years ago, when the present marital blood-test legislation was passed. He presented the senior medical officer in Kittery with a set of applications for the Board of Registration in Medicine, for the reason that a license from the Board of Registration in Medicine is required in the people who certify for marriage purposes. The senior medical officer told Dr. Burroughs that the Navy was considering following the precedent already established by the Surgeon General of the Army, namely, that medical officers of the Army were prohibited from engaging in civilian practice in the vicinity of the Army posts. No such regulation had been issued by the Surgeon General of the Navy, but it was anticipated that such a regulation might be issued, and in the meantime, the Surgeon General's policy was definitely to discourage such activity on the part of the naval officers. The medical officer returned the set of blank applications with the comment that the Navy doctors had no use for them.

Dr. Burroughs further stated that the Attorney General had informally ruled that, since the Navy Yard in Kittery is actually located on the borders of New Hampshire, on the line of New Hampshire and Maine, a commission in the Navy should be regarded as having the same effect as a license in the adjoining state; hence, a naval medical officer situated at Portsmouth, holding a commission from the Navy, should be considered authorized to practice medicine in New Hampshire, under that exception in Section 18 of the Medical Practice Act, which states that so long as he does not establish an office or appoint a place to meet patients in New Hampshire, any physician practicing on the borders of New Hampshire and duly licensed in his own jurisdiction does not require a license in New Hampshire.

Dr. Gage stated that the Belknap County Medical Society requested the New Hampshire Medical Society to grant to Edwin P. Hodsdon, of Lakeport, the privilege of affiliate membership with the Society, and the privilege of nonpayment of dues. Dr. Hodsdon was given a gold medal for fifty years of practice at this annual meeting.

Dr. H. O. Smith moved that Dr. Hodsdon be made an affiliate member of the New Hampshire Medical Society.

This motion was duly seconded and was carried.

The first meeting of the House of Delegates was adjourned at 10:55 p.m.

* * *

The House of Delegates convened at the Hotel Carpenter, Manchester, on May 12, 1942, at 8:30 a.m., with Speaker Timothy F. Rock, of Nashua, presiding.

The following members answered the roll call:

The President, *ex-officio*
The Vice-President, *ex-officio*
The Secretary-Treasurer, *ex-officio*
P. R. Hoyt, Laconia (alternate for Lyall A. Middleton, Plymouth)
Earl J. Gage, Laconia (alternate for Richard W. Robinson, Laconia)
W. J. Paul Dye, Wolfeboro
Francis J. C. Dube, Center Ossipee
Walter F. Taylor, Keene
Norris H. Robertson, Keene
Leander P. Beaudoin, Berlin
Lewis C. Aldrich, Jefferson
Everett C. Campbell, Woodsville
Arthur W. Burnham, Lebanon
Leslie K. Sycamore, Hanover
George V. Fiske, Manchester
Deering G. Smith, Nashua
H. O. Smith, Hudson (appointed as alternate for Charles H. Cutler, Peterborough)
George C. Wilkins, Manchester (appointed as alternate for Clarence E. Dunbar, Manchester)
William P. Clough, Jr., New London
Clinton R. Mullins, Concord
Frederick S. Gray, Portsmouth
Anthony E. Peters, Portsmouth
Louis C. Theobald, Exeter
Albert E. Barcomb, Farmington
George G. McGregor, Durham
Bernard P. Haubrich, Claremont
Addison Roe, Newport

The Speaker declared a quorum present, and asked for further reports of standing committees.

Dr. Sycamore repeated the recommendations of the Committee on Medical Economics. Dr. Metcalf inquired what was meant by the words, "to replace," in the report.

Dr. Sycamore answered that, if a physician goes into a town because somebody else has gone out, such a residence is not necessarily a definite arrangement. The recommendations present one possibility of trying to protect the man who leaves his practice.

Dr. Dye stated that a ruling from the Attorney General's office concerning such limited licensure would be required. Under the present law, there is no provision for making any type of limited licensure. Dr. Dye also said that at present the Medical Practice Act requires that a man be a citizen of the United States to practice and to receive a license in New Hampshire. About the only type of person to whom a limited license could be given would be a refugee physician. In Dr. Dye's opinion, it would be very difficult and impractical to give any qualified physician a limited license to practice in New Hampshire. In other words, if a man is a graduate of a first-class medical school, having had two years preliminary education in a college approved by the Board of Registration in Medicine and one year's internship, and has passed the examinations, there would be no way of limiting his license. One disadvantageous point is that a refugee physician, if granted a limited license, might go into a community, and it is a well-known fact that every physician who practices in any community builds up a certain amount of following, so that after a year or two of practicing in that community, there would always be a group of people who would bring pressure to bear that his license be retained and that he be privileged to continue to practice there.

Dr. Sycamore stated that the Committee on Medical Economics was thinking of the refugee-physician problem, in presenting this subject for discussion. It was in an attempt to anticipate such pressure that it was deemed advisable to discuss the problem.

Dr. Wilkins asked whether this question would not finally come down to an agreement between the officials of the Procurement and Assignment Service in the State and the State Board of Registration in Medicine.

Dr. D. G. Smith was of the opinion that the resolution does not apply only to refugee physicians: there is a great possibility that it may be necessary to transplant doctors from the larger cities,—for example, from Massachusetts to New Hampshire,—to take care of some of the rural areas. He agreed that the matter should be taken care of by the Board of Registration in Medicine, under the war emergency clause and the present statutes. He believed that the committee's idea was to obtain an opinion from the House of Delegates regarding whether or not they would approve limited licensure; such a statement would help the Board of Registration in Medicine somewhat in solving this problem.

Dr. Metcalf asked how, under the present law, a limited license could be issued.

Dr. D. G. Smith replied that the Board of Registration in Medicine would practically dictate it, under a war clause. He pointed to a section in the by-laws that requires applicants for examination for licensure to meet certain conditions: two years of premedical training, graduation from an approved school, a year's internship, citizenship and so on; the last part of that section reads: "The provisions of this section may be suspended in whole or in part by order of the Board on account of war or other threatened or existing national calamity."

So that, in the final analysis, the Board may license anybody it wishes to license. The position of the Board up to the present time has been not to let down the bars in any particular case—so far as letting in men from substandard schools and men who have not had any premedical education is concerned.

Dr. Taylor moved that the House of Delegates approve the limited license, in principle.

This motion was duly seconded and was carried, with one dissenting vote.

Dr. Dye, reporting for the Committee on Amendments to the Constitution and By-Laws, stated that various matters, largely of a technical nature, were referred to the committee. It was recommended that Chapter VIII, Section 6, of the by-laws be amended to read in full as follows: "Section 6. The Committee on Medical Economics shall consist of three members." Previously, this particular section read: "Section 6. The Committee on Medical Economics shall consist of three members, one of whom shall be elected annually for a term of three years."

This change was suggested to make it a standing committee and not a rotating committee.

Dr. Dye moved the adoption of the proposed change.

This motion was duly seconded and was carried.

Further changes recommended were as follows:

We recommend that Chapter VIII, Section 10, be amended to read as follows: "The Committee on Mental Hygiene shall consist of three members, whose duties shall be to study this special subject, co-operate as far as possible with the heads of the state institu-

tions under medical supervision and report to the society."

The only change is in the striking out of the words "Social Hygiene" and changing the plural to the singular.

I move the adoption of this amendment to the by-laws, regarding Section 10 of Chapter VIII.

In Section 12, it was a technical matter, to make this a standing committee instead of a rotating committee.

We recommend that this section be amended to read as follows: "The Committee on Medical Education and Hospitals shall consist of three members."

I recommend and I move a change in the heading of Section 1, Chapter VIII, naming one Committee as the Committee on Mental Hygiene, omitting "and Social."

With reference to Chapter I, Section 5, regarding membership, this matter was brought up because Dr. Hodsdon, who was a member of the New Hampshire Medical Society, without being a member of a component medical society, became a member prior to the formation of the county societies and now wishes to be made an affiliate member.

I should like to read the proposed amendment to Section 5 completely, as follows: "Any physician who has been a member of this Society for a continuous term of fifteen years and is either not less than sixty-five years of age or totally disabled, on personal application or on the request of his county society, may be made an affiliate member by a majority vote of the House of Delegates. Affiliate members shall have the same rights and privileges as other members of the Society, but shall not be required to pay dues."

Unless some one has any other particular question in mind regarding members, that completes our report on amendments to the by-laws.

We were asked to give an opinion regarding the clarification of the membership qualifications. I shall first read Section 2, of Article IV, of the Constitution, which has to do with the composition of the society: "The members of the Society shall be the members of the component county medical societies and the present members of the New Hampshire Medical Society."

In the first place, there is no specification that residence in New Hampshire is necessary for membership. Secondly, men who were not previously members of the Society prior to the formation of the county medical societies must now be members of a component county medical society before being accepted by the State Society.

Dr. Sanders, referring to the question of eligibility to membership in a county medical society, stated that Dr. Amsden, of Ackworth, had come to New Hampshire from New York and had applied for membership in the New Hampshire Medical Society. He continued:

I found, in looking up the matter, that it was required that a member be licensed in New Hampshire and that he be in active practice. Those requirements, I think, come through the constitution and by-laws that each county society is supposed to adopt on recommendation of the New Hampshire Medical Society.

I wondered if that was the regular practice of county societies in admitting members to the county membership — that is, a man could not merely reside, and not be in active practice or licensed, in the State, and still be a member of a county medical society.

Dr. Amsden took out a New Hampshire license, and is in practice in the State. He is a retired psychiatrist, and I think perhaps did not intend to practice in the State; however, he is practicing somewhat to keep up his membership in the American Medical Association. He gave up his New York State membership, and had to have some county membership to keep up the American Medical Association membership.

Dr. D. G. Smith moved that Article XII be deleted from the Constitution, explaining that such an action must be proposed at one annual session and then acted on at the succeeding annual session.

This motion was duly seconded and was carried.

Dr. Wilkins moved that Article XIII of the Constitution be made Article XII.

This motion was duly seconded and was carried.

Dr. Dye moved that since the by-laws had been amended to the effect that a present member of the Society may apply for affiliate membership, Dr. Hodsdon, who had written to the Society, be made an affiliate member of the New Hampshire Medical Society.

This motion was duly seconded and was carried.

Dr. Dye stated that this was not in the nature of a motion and that the delegates might want to discuss the question. He referred to Dr. J. Dellinger Barney, who had relinquished his residence in New Hampshire and wondered what his membership status would be. So far as the committee could determine, he could follow one of two courses. If his county society retained his membership, Dr. Barney could continue to be a member of the Society, since there is no provision in the constitution of the Society that a man must be a resident of the State to be a member of the Society. Should he not continue as a member of his county society, by virtue of the local county constitution, under Section 4, Article IV, he could be proposed by the House of Delegates as an honorary member of the Society.

Dr. D. G. Smith cautioned the Committee on Constitution and By-Laws to be sure that it did not conflict with the constitution of the Cheshire County Medical Society and with the constitution and by-laws of the American Medical Association, since he believed that such a conflict might occur:

The model constitution of the county society says that a doctor, to be eligible for membership, must reside and practice in the county in which he is applying for membership, with the exception that men on county lines and state lines in adjoining counties or states can belong to that county society.

Dr. Robertson inquired about members who practice in New Hampshire and are members in Massachusetts and have kept up their Massachusetts membership through the years.

Dr. D. G. Smith answered that, strictly speaking, such members can belong only to one county and one state medical society.

Dr. Sycamore said that there should be a general ruling that would cover these cases and that, in his opinion, a simpler solution would be to make them honorary members; the question of a conflict with the American Medical Association rulings would then be avoided.

Dr. Dye moved that any physician who is not a resident of New Hampshire have his individual status regarding membership in the New Hampshire Medical Society, subject to the recommendation of his component county medical society.

Dr. Sycamore asked if the American Medical Association or the component societies had the final authority.

Dr. Rock declared that he did not know who had the final authority.

Dr. Dye stated that, apparently, certain members of the House of Delegates wanted a ruling regarding cases like that of Dr. Barney, and that he had therefore moved that each county society decide the matter.

Dr. D. G. Smith stated that the question involved a double representation: a man belongs to one county society, and his membership counts as a quota toward the state society in electing a delegate; the same thing would be true if he belonged to two county societies in the State. His name would be added to their membership lists, making a quota for the members necessary to have an additional delegate to the House of Delegates. In the American Medical Association, he is listed in two different states; therefore, he contributes to New Hampshire's quota for a delegate and he contributes to the Massachusetts quota.

After further discussion, the motion previously made by Dr. Dye was seconded by Dr. Fiske and was carried.

The second meeting of the House of Delegates was adjourned at 10 a.m.

* * *

The House of Delegates convened at the Hotel Carpenter, Manchester, on May 13, 1942, at 8:30 a.m., with Speaker Timothy F. Rock, of Nashua, presiding.

The following members answered the roll call:

The President, *ex-officio*
The Vice-President, *ex-officio*
The Secretary-Treasurer, *ex-officio*
Earl J. Gage, Laconia (alternate for Richard W. Robinson, Laconia)
W. J. Paul Dye, Wolfeboro
Francis J. C. Dube, Center Ossipee
Lewis C. Aldrich, Jefferson
Everett C. Campbell, Woodsville
George V. Fiske, Manchester
Deering G. Smith, Nashua
H. O. Smith, Hudson (appointed as alternate for Charles H. Cutler, Peterborough)
George G. Wilkins, Manchester (appointed as alternate for Clarence E. Dunbar, Manchester)
William P. Clough, Jr., New London
Frederick S. Gray, Portsmouth
Albert E. Barcomb, Farmington
Bernard P. Haubrich, Claremont
Addison Roe, Newport

Dr. Dye reported for the Committee on Nominations, as follows:

OFFICERS

President: Timothy F. Rock, Park R. Hoyt and Arthur W. Burnham.

Vice-President: Ralph W. Tuttle, James W. Jameson and Fred Fernald.

Councilor for Coos County (five-year term): Richard E. Wilder.

Councilor for Carroll County (five-year term): W. J. Paul Dye.

Trustee emeritus: Henry O. Smith.

Trustee for three years: Clarence O. Coburn.

House of Delegates: speaker, Charles H. Parsons; vice-speaker, George F. Dwinell.

Necrologist: Henry H. Amsden.

Delegate (American Medical Association): Deering G. Smith.

Alternate Delegate (American Medical Association): Emery M. Fitch.

Delegates to New England Societies (1942):

Maine: Leopold T. Togus, Lawrence R. Hazzard.
Vermont: Harry B. Carpenter.
Massachusetts: Wallis D. Walker, W. J. Paul Dye.
Rhode Island: Reginald F. DeWitt, Ellsworth M. Tracy.
Connecticut: John F. Gile, James E. Bovaird.

STANDING COMMITTEES

Amendments to Constitution and By-Laws: James B. Woodman, W. J. Paul Dye, Frederick S. Gray.

Child Health: Colin C. Stewart, Jr., Travis P. Burroughs, Franklin N. Rogers.

Control of Cancer: George C. Wilkins, Ralph E. Miller, George F. Dwinell.

Maternity and Infancy: Robert O. Blood, Benjamin P. Burpee, Marion Fairfield.

Medical Economics: Leslie K. Sycamore, Richard W. Robinson, Francis J. C. Dube.

Medical Education and Hospitals: John P. Bowler, James W. Jameson, Herbert L. Taylor.

Mental Hygiene: Charles H. Dolloff, Benjamin W. Baker, John B. McKenna.

Public Health: Travis P. Burroughs, Anthony E. Peters, Harris E. Powers.

Public Relations: The President, The Vice-President, The Secretary-Treasurer, Robert J. Graves, Ezra A. Jones.

Publication: Carleton R. Metcalf, John F. Gile, Emery M. Fitch.

Scientific Work: Carleton R. Metcalf, Frederick P. Scribner, Sven Gundersen.

Tuberculosis: Robert B. Kerr, M. Dawson Tyson, Clarence O. Coburn.

Medical Preparedness: Deering G. Smith, Ezra A. Jones, Carleton R. Metcalf.

The report was accepted. Dr. Timothy F. Rock, of Nashua, was elected president.

Dr. James W. Jameson, of Concord, was elected vice-president.

Dr. Dye, for the Committee on Nominations, recommended a standing vote on one other nomination on the slate—for trustee emeritus, Dr. Henry O. Smith, of Hudson.

The Secretary was instructed to cast one vote for Dr. Henry O. Smith as trustee emeritus; this was done, and he was declared duly elected.

The Secretary was instructed to cast one ballot for the remainder of officers and committee members as nominated; this was done, and all were declared duly elected.

Dr. Metcalf moved a vote of gratitude to the members in Manchester who had arranged the details of the meeting; it was so voted.

Dr. Wilkins then presented the report of the Committee on the Control of Cancer.

Report of the Committee on the Control of Cancer

The Committee on the Control of Cancer has sent to every physician in New Hampshire the usual three letters of information regarding specific phases of cancer diagnosis or treatment. The first letter was devoted to prostatic carcinoma, a condition more frequently found than is generally realized; the second briefly discussed diagnosis and treatment of cancer of the mouth; and the last called attention to the valuable lay educational work performed by the Women's Field Army, stressed the value of urging people to have periodic physical examinations and urged more education of the public in recognizing the significance of irregular uterine bleeding, as well as the urgency for immediate thorough examination in the event of rectal bleeding. Too many physicians are still too reluctant in carrying out the necessary examinations required to determine the cause of bleeding in these two conditions.

During the year, requests for full sets of these letters, sent since 1934 to New Hampshire physicians, have been received from cancer committees in Maine, Vermont, Kansas and Georgia, and from the American Society for the Control of Cancer.

In 1935, at the request of the Cancer Commission, the State Board of Health made cancer a reportable disease. The physicians of the State and the hospitals were appealed to by both the commission and the Board of Health to report all cases of cancer coming to their attention. The Pathological Laboratory at Hanover co-operated to the extent of mailing to physicians a partly filled out form with each positive cancer report. In 1941, the Board of Health, at the suggestion of the commission, repealed this ruling because, during the six years that cancer was reportable, it was found that there were reported only about 50 per cent as many cancer cases as there were cancer deaths and the reported cases came almost entirely from the cancer clinics. The Cancer Commission suggested this repeal because it seemed fruitless to continue an effort when such a lack of co-operation on the part of physicians was evidenced. Without full co-operation, there is no way of ascertaining how many active cancer cases there are in the State.

The diagnostic clinics and treatment centers supervised by the Cancer Commission have continued their excellent work, and about sixty members of the New Hampshire Medical Society are regularly taking active part in this clinic service. There is still much evidence of ignorance, apathy and delay on the part of patients in spite of the efforts of the medical profession and the widespread distribution of small pamphlets carried each year into every city, town and village by the Women's Field Army. Last year, ninety-five thousand pieces of literature were distributed, and the New Hampshire leaflets are now printed in English, French, Polish and Greek. We urge all physicians of the State to read the reference book on cancer, which was sent to every physician in the State in 1940 by the Women's Field Army.

In Massachusetts, the intensive cancer-control campaign took ten years of constant effort before the mortality rate dropped definitely downward. The New Hampshire program is now in its seventh year, and we hope soon to observe tangible evidence of the effectiveness of medical and lay education, combined with improved surgical and radiologic treatment.

Much credit should go to the alertness of the majority of the physicians in the State for their keenness and ability in recognizing cancer early and inducing patients to seek early treatment. A minority of physicians unfortunately still have a lackadaisical attitude toward thorough and adequate physical examination, early indications of cancer and the methods of making early diagnosis. These men need education, but they are probably the ones who throw our letters into the wastebasket unread.

Your committee has spent $44.35 for printing, stationery and postage in sending out nearly two thousand letters, the balance of $5.65 having been returned to the treasurer. We request an appropriation of $50.00 for the coming year.

GEORGE C. WILKINS, *Chairman*
HOWARD N. KINGSFORD
GEORGE F. DWINELL

Dr. Sycamore, for the Committee on Officers' Reports, recommended that the requested appropriation of $50.00 be approved.

This motion was duly seconded and was carried.

The report of the Committee on Medical Education and Hospitals was then presented.

Report of the Committee on Medical Education and Hospitals

POSTGRADUATE MEDICAL EDUCATION

The fifth annual meeting of the Associated State Postgraduate Committees was held in Cleveland in 1941 in conjunction with the meeting of the American Medical Association. The meeting of this committee was attended by Dr. Deering G. Smith as our representative. The new chairman is Dr. Frank R. Ober, of Boston. The New England member of the Executive Committee is Dr. Frederick T. Hill, of Waterville, Maine.

This committee has been working for some five years in the attempt to co-ordinate the activities of the various state committees on medical education and hospitals, and the program being assumed by them is a steadily more pretentious one. In this connection, at their last meeting, it was voted that an assessment of $50.00 be made against each state to provide funds for the activities of this committee. It is the opinion of Dr. Smith, who attended this meeting, and of several others, that the activities of postgraduate education in the State are too limited and will without doubt be more limited in the near future to warrant this expenditure by the Society at this time. This reason leaves out altogether the question whether or not such assessment should be on the basis of population rather than a fixed, uniform figure. It is therefore the belief of this committee that the State assessment be not recommended to the Society at this particular time.

COMMONWEALTH FUND FELLOWSHIPS

A year ago, we rendered a brief review of the activities of the Commonwealth Fund Fellowships in this state for the preceding five years through which they had been operating. It is to be expected under present conditions that there has been less interest in this activity this year as measured by the response to the circularization of the membership of the New Hampshire Medical Society. To date, there have been requests for application blanks from only eight members of the Society, and appointments have not yet been announced by the Commonwealth Fund.

There seems to be little more that can be done in the way of calling the attention of the membership to these fellowships, which seem to be so generous and intelligent in their conception, and it is to be hoped that their value will not be lost sight of in the exigencies of the times in which we are now living.

JOHN P. BOWLER, *Chairman*
JAMES W. JAMESON
HERBERT L. TAYLOR

Dr. Amsden read the report of the necrologist.

Report of the Necrologist

The following deaths have occurred since the last annual meeting:

NAME	ADDRESS	DATE DECEASED
Abbott, Charles B.	Penacook	March 24, 1941
Cheever, Nathaniel F.	Greenfield	June 15, 1941
Cobb, Joseph J.	Berlin	July 15, 1941
Daudelin, Alfred	Nashua	September 21, 1941
Deming, Robert M.	Glencliff	January 28, 1942
Hinds, Wm. H. W., Jr.	Milford	March 10, 1942
Kittredge, Frank E.	Nashua	July 8, 1941
LaFrance, Albert J.	Laconia	March 12, 1942
Lamb, Zenas F.	Keene	April 28, 1941
Marks, Homer H.	Berlin	April 11, 1941
Noyes, Walter F.	Colebrook	May 24, 1941
Potvin, Victor E.	Claremont	December 10, 1941
Sanborn, Benjamin E.	Manchester	October 3, 1941
Webber, Norman B.	Manchester	February 22, 1942
Weinberg, Philip B.	Whitefield	August 27, 1941

HENRY H. AMSDEN

Dr. Rock expressed appreciation to the members of the House of Delegates for their assistance in carrying on these meetings.

The third and final meeting of the House of Delegates was adjourned at 9:20 a.m.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 28341

PRESENTATION OF CASE

A fifty-year-old man was admitted to the hospital because of cough and fever.

During the week prior to admission, the patient suffered with a "head and chest" cold characterized by a great deal of coughing productive of small amounts (not over a teaspoonful a day) of whitish sputum. He was seen by his physician on the evening prior to entering the hospital. At this time, he was coughing and vomiting, and there was some right-lower-quadrant pain associated with the bouts of coughing. There was no hemoptysis, and no chills or chest pain. Rales were audible at the posterior base of the left lung, and a murmur, believed to be early diastolic, was heard at the apex. A urine specimen was said to have contained many blood cells and much albumin.

The family history was irrelevant.

The past history was unremarkable except for an accident that occurred one year previously, when the patient fell from a truck and sustained a fractured skull. He was unconscious for twelve days, and since then he had suffered with severe left frontal headaches, dizziness and tinnitus.

Physical examination revealed a well-developed man in no distress who coughed frequently, bringing up a thin, white sputum. The chest was barrel shaped. Examination of the heart was negative, although one observer reported a systolic murmur audible to the left of the sternum and a sharp pulmonic sound. There was some impairment of resonance at both lung bases posteriorly. The percussion note was hyperresonant, with some dullness over the left lower lobe posteriorly. Sticky rales were audible in the left chest, most marked at the bases. A few persistent inspiratory rales were audible in the left infraclavicular region anteriorly. There was deep tenderness in the right upper quadrant, but no abdominal masses were felt. There was also left costovertebral tenderness. There was prominent clubbing of the fingers.

The blood pressure was 138 systolic, 80 diastolic. The temperature was 100.6°F., the pulse 96 and the respirations 24.

Examination of the blood revealed a hemoglobin of 10.7 gm., and a white-cell count of 8600 with 67 per cent polymorphonuclears, 5 per cent large lymphocytes, 12 per cent small lymphocytes and 16 per cent monocytes. The blood Hinton test was reported doubtful and, when repeated, was unsatisfactory. The nonprotein nitrogen was 60 mg. per 100 cc. Examination of the urine revealed a specific gravity of 1.012, a +++ test for albumin and 100 red blood cells, 10 white blood cells and 10 epithelial cells per high-power field. No acid-fast bacilli or other pathogenic organisms were found in the sputum. A blood culture was negative.

X-ray examination of the chest showed symmetrical and diffuse linear and hazy density of both lungs, more marked at the bases. Both lung roots were enlarged, and the right side of the supracardiac shadow was moderately prominent. The heart was not definitely enlarged but was triangular — prominent across the base. The costophrenic angles were hazy, particularly in the posterior portion.

The patient complained of fever and sweating during the entire period of hospitalization. There was no chest pain, and no change in the amount of coughing. Repeated urinary examinations revealed a persistently low specific gravity, a +++ test for albumin, rare hyaline and cellular casts, many white blood cells and large numbers of red blood cells. The urine cultures revealed no growth.

Intravenous pyelograms showed no excretion of the dye. Small areas of dense calcification were found in the region of the upper pole of the right kidney. There were small flecks of calcification in the right pelvis close to the sacroiliac joint. The kidneys were normal in size and shape. The liver edge was lower than normal, and a soft-tissue mass, apparently the spleen, overlay the upper pole of the left kidney.

On the fifth day, there was less moisture in the lungs. The nonprotein nitrogen had risen to 111 mg. per 100 cc. two days later. On the ninth hospital day, while reaching for a bedpan, the patient suddenly noted pain in the right hand. The hand became cold, and the patient could not dorsiflex it. No brachial or radial pulse was felt; there were faint arterial pulsations in the axilla. On the following day, the pain was somewhat relieved. The radial pulse gradually returned. The blood pressure on this day was 82 systolic, 72 diastolic, in the right arm and 130 systolic, 70 diastolic, in the left.

A Congo-red test revealed 70 per cent retention of the dye in the serum after an hour.

On the thirteenth day, the spleen was felt two or three fingerbreadths below the costal margin. The strength in the right hand had improved considerably. An x-ray film of the chest showed

little change. The diffuse, hazy density appeared to have cleared slightly.

Two and a half weeks after admission, the patient complained of slight pain in the left shoulder, which became progressively worse, particularly when he took a deep breath. A faint rub synchronous with the heart beat was audible over the entire precordium. The heart sounds were distant, but the radial pulse was regular and strong. The blood pressure was 140 systolic, 85 diastolic. That evening while on a bedpan, the patient suddenly began to breathe stertorously and expired.

Differential Diagnosis

Dr. Wyman Richardson: It seems very clear to me that this patient had a bacterial endocarditis, that he had multiple emboli with infarcts, and that he had some type of renal failure, all of which led to sudden death. The questions to be answered are: What type of endocarditis was it? What type of renal failure? What was the cause of the apparent pericarditis? What was the immediate cause of death? I should like to have Dr. Hampton tell me, if he will or can, whether the x-ray film of the heart shows any suggestion of previous long-standing heart disease, perhaps congenital heart disease or some other type.

Dr. Aubrey O. Hampton: I think the heart is definitely enlarged. It is triangular and the curve of the right auricle is high. The aorta is small, and the pulmonary vessels are dilated. The thing that bothers me most is whether there are also enlarged lymph nodes at both lung roots aside from the dilated pulmonary vessels. The heart has more the appearance of mitral disease than congenital heart disease. There is no fluoroscopic note of how large the left auricle was. This is the lateral view, but there is no oblique view, which would help more than the lateral. This shadow to the right of the mediastinum is longer and lower than the usual azygos node that one sees in sarcoid and lymphoma. It could be the vena cava, I suppose, but there must be something in addition to the vena cava there. When one sees the enlarged spleen, one wonders if these enlarged nodes are associated with that — provided they *are* enlarged nodes. I am not too happy about it. There is also what appears to be edema of the lung and, in addition, diffuse linear density in the lung, which could be connected with enlarged lymph nodes, as in sarcoid. I think that there was a definite heart lesion, and that there probably were enlarged lymph nodes.

Dr. Richardson: What about this?

Dr. Hampton: That is a focus of calcification, but I do not know where it is.

Dr. Richardson: I was not paying too much attention to it.

Dr. Hampton: It shifts in relation to the kidney cortex. Here it is about 2.5 cm. from the margin, and there is 1.5 cm. I assume that it is outside the kidney. It could be in the gall bladder, but it does not look like either gallstone or renal stone. The kidneys show very poor function. This is a 30-minute film with intravenous dye, and as you see, there is nothing in the bladder. The right kidney is low, and the edge of the liver is not so low as the edge of the spleen.

Dr. Harwood: Could the heart shadow be simple dilatation?

Dr. Hampton: I do not believe so. It has an abnormal shape. Simple dilatation would maintain a normal shape, I think.

Dr. Richardson: I shall disregard the calcification, which is a dangerous thing to do with all these calcium experts around. There is x-ray evidence that there may have been a preceding heart lesion, and the picture is somewhat suggestive of the possibility of mitral stenosis, if I understand Dr. Hampton correctly. That would give at least a background for subacute bacterial endocarditis. I was prepared to make this diagnosis in the absence of evidence of preceding heart disease because I thought that the picture was very clear. Assuming for the moment that I am right about it, if this was bacterial endocarditis, what organism was involved? It seems to me that one of the acute types — a beta-hemolytic streptococcus or pneumococcus — would have grown out in a culture. If the blood picture is correctly reported, the presence of an increased number of monocytes without a definite polymorphonuclear formula is suggestive that this was not an acute type, but more likely one due to the alpha-hemolytic streptococcus. I believe that this patient had bacterial endocarditis, with vegetations on the valves infected by the *Streptococcus viridans*.

So far as the type of renal failure is concerned, we know, accepting that diagnosis, that nephritis is a common complication of bacterial endocarditis, and I believe something like 90 per cent are glomerular and 10 per cent embolic; therefore, one ought to be in favor of the glomerular type. However, certain things lead me to suspect that the patient had embolic nephritis, or even large renal infarcts. One is that he had costovertebral tenderness, which is not very definite evidence. Another is that he had evidence of emboli in the systemic circulation. There was an embolus in the blood vessels of the arm, and it seems to me that there was enough suggestion of emboli for embolic nephritis to be considered; it is even quite possible that the patient had large renal infarcts. I therefore think that is so, and that he had uremia. I believe the uremia caused the pericardial friction rub. I do not know about that sequence of

events. I suppose that pericarditis can be associated with reactivation of rheumatic heart disease, but I think one might reasonably see that this patient had uremic pericarditis.

The cause of death seems to me most likely embolic, and since many of the embolic phenomena have been in the systemic circulation, it seems more likely that the patient had a cerebral embolus than a pulmonary embolus. I wondered whether this could have been some type of cancer and whether all these phenomena were due to cardiac invasion, possibly with the production of thrombus and embolus. There is a great deal going on in the chest that Dr. Hampton does not like. I do not like it either, but I shall not change my original diagnosis on the basis of the x-ray films. I shall say that there was not any type of malignant tumor. I do not know what the calcification was. If the patient had had a previous infarct, he might have had calcium in the infarct. In bacterial endocarditis, infarction is very common. One would expect infarcts in many organs, especially the lungs, spleen and kidneys, and peripheral infarcts elsewhere. Therefore, I make a diagnosis of subacute bacterial endocarditis, probably engrafted on chronic rheumatic heart disease, alpha-hemolytic streptococcus infection and embolic nephritis, probably with large renal infarcts, uremic pericarditis, and a large cerebral embolus as the immediate cause of death.

DR. FULLER ALBRIGHT: Are you disturbed that the kidneys were small? They are usually not small with large fresh infarcts.

DR. RICHARDSON: I think it is very reasonable. I think there was endocarditis for some time before the three weeks mentioned in the history.

DR. ALBRIGHT: How about the sterile urine? Embolic nephritis is not associated with sterile urine.

DR. RICHARDSON: I do not know. I do not believe that is necessarily true.

DR. ALBRIGHT: I do not know that it is, either, as a matter of fact.

DR. F. DENNETTE ADAMS: Why do you think that a large cerebral embolus, rather than a pulmonary embolus, accounted for this very sudden death? Do you not expect cerebral hemorrhage to last a few hours?

DR. RICHARDSON: Yes; my reasoning was that there was one in the arm, and it seemed more on the systemic than the pulmonary side. Accordingly, I put it there. I thought the description was not quite that of ordinary pulmonary embolism, but there is really nothing to go on.

DR. MAURICE FREMONT-SMITH: Is the x-ray film consistent with pericardial fluid?

DR. HAMPTON: With the exception of the lung roots, yes. If the patient had perfectly normal lungs and pulmonary vascular shadows, it would be quite consistent with it, but apparently he had something going on in the pulmonary vascular system for a long time before that. The heart is not very large, so that the whole thing can be explained by mitral stenosis.

DR. ALBRIGHT: You do not know the calcium and serum protein?

DR. TRACY B. MALLORY: No.

DR. HAMPTON: Another thing — the chest films were not taken at the time he had pericardial symptoms.

DR. WILLIAM BISHOP: I should like to comment about the head injury a year previously. I saw the patient outside the hospital for quite a long time because of that trouble, and had a chance to examine him repeatedly. I sent him to Dr. John S. Hodgson, with the question of whether he had residual hematoma following the head injury and fracture, but we could not make out that there were any definite signs. He had no good localizing evidence of any really organic cerebral damage, but it seemed to me as I observed him that he was slower mentally than originally, although he was never very brilliant, and it was difficult to be sure of that. He was asked whether or not he felt able to go to work. At times, he thought he could, and at other times that he could not; but he never did return to any occupation. His chief complaints up to the time of the last illness were headache and dizziness, dizziness especially on looking up too quickly. I saw him just before admission to the hospital, and the heart murmur, rales and especially the markedly bloody urine were the things that led me to urge him to come to the hospital. I was more or less jockeyed out of my impression that he had a heart murmur. Some heard it, and some did not. Then I changed my mind and said it was a systolic murmur. Now I should like to think I really heard a diastolic murmur. I knew that there must have been something abnormal in the heart that was giving rise to emboli, perhaps a mural thrombus. I did not come to a definite conclusion that he had bacterial endocarditis.

DR. FLETCHER H. COLBY: I saw the man mainly because of the hematuria. The gross hematuria in conjunction with the high nonprotein nitrogen and lack of excretion of dye in the intravenous pyelogram suggested some sort of nephritis. On the other hand, when I first saw him the nonprotein nitrogen was 50 or 60 mg. per 100 cc., and we planned to cystoscope him to see if we could discover some source of bleeding, such as a bladder tumor. He quickly got too ill, and we canceled arrangements. That was the time of the embolus to the axillary artery. The spleen was easily felt when he first came in. That was not brought out in the history.

Clinical Diagnoses

Acute glomerulonephritis.
Uremia.

Dr. Richardson's Diagnoses

Subacute bacterial endocarditis (alpha-hemolytic streptococcus), probably secondary to chronic rheumatic heart disease.
Embolic nephritis, with large renal infarcts.
Uremic pericarditis.
Cerebral embolus.
Multiple infarcts.

Anatomical Diagnoses

Endocarditis, subacute bacterial, mitral.
Endocarditis, chronic rheumatic, mitral, with stenosis, and aortic, with interadherence of cusps.
Cardiac hypertrophy, moderate.
Pericarditis, serofibrinous, early.
Subacute glomerular nephritis.
Pulmonary hemorrhage, bilateral, interstitial, focal.
Splenic infarction.
Embolism of brachial artery, with extension into subcapsular artery.
Encephalomalacia, localized, bilateral, post-traumatic (head injury).
Hypertrophic pulmonary osteoarthropathy.

Pathological Discussion

Dr. Mallory: Post-mortem examination showed bacterial vegetations on the mitral valve, superimposed on a well-marked calcareous rheumatic stenosis. There had been numerous emboli from this source. We located the one in the brachial artery, and found two small infarcts in the spleen and two in the kidney. The kidneys were very large, weighing 600 gm. The cortices were markedly swollen, and the surfaces were smooth but covered with petechial hemorrhages. There has been quite a little discussion of the question of embolic nephritis, and I think the clinicians today have used the term in a sense that is distinctly different from the usage of most pathologists. In the average case of bacterial endocarditis, one finds some changes in the glomeruli, and patients have during life, at least at times, hematuria. The ordinary case of bacterial endocarditis, however, shows these glomerular changes only in isolated glomeruli, whereas the majority of the glomeruli remain normal. Moreover, even an involved glomerulus may show inflammatory changes limited to only a portion of the tuft, the other loops of the tuft remaining perfectly normal. Consequently, most of the patients have good renal function, and a uremic death is rare. Because this type of glomerular disease occurs so regularly in bacterial endocarditis, it has often been called "embolic nephritis." There is really in most cases no direct evidence that it is embolic, and the same lesion is frequently found in acute disseminated lupus without endocarditis or other focus from which embolism could arise. Therefore, the term "focal glomerular nephritis" is preferable. I think what Dr. Richardson and Dr. Albright have been referring to is true demonstrable embolism to the kidney. This may be so massive, or miliary emboli may be so numerous, that renal insufficiency develops. Such cases are, however, rare. In this case, the microscopic sections showed a true diffuse glomerular nephritis indistinguishable from a primary glomerulonephritis. All glomeruli were involved more or less uniformly and completely, so that the development of renal insufficiency is not surprising.

There were one or two other findings of interest. The myocardium showed very obvious fibrous scarring in scattered spots throughout the heart. The aorta and coronary arteries, however, were practically free from sclerosis, and there was no evidence of any emboli to the coronary arteries. Later, on microscopic examination, it was apparent that these spots of fibrosis in the myocardium were all extremely old. There were no signs of any fresh myocardial necrosis, and no trace of inflammatory reaction in any of these scars. Consequently, this myocarditis must be put far into the past history, perhaps back to the time when the patient acquired the mitral stenosis, and hence may well have been of rheumatic origin. It is also conceivable that he had had diphtheria unknown to us in the past, which is the commonest source of that sort of lesion. The brain did show definite evidence of damage from the accident a year before. There were foci of superficial cortical atrophy, with marked hemosiderosis of the overlying arachnoid.

Dr. Hampton: Sudden death was due to what?

Dr. Mallory: It was due to no cause that we could discover. There was massive acute pulmonary hemorrhage, or edema — very hemorrhagic serosanguineous fluid. The heart weighed 500 gm., and there was no atheroma of either the pulmonary or the coronary arteries.

CASE 28342

Presentation of Case

A seventy-year-old man was admitted to the hospital because of persistent hematuria.

Three months before admission, the patient slipped while walking and twisted his right side. Following this, he had slight pain and an ache in the right flank that disappeared after several days.

At that time, he noticed the onset of hematuria, persistent to the day of admission except for a single period of three weeks. The degree of hematuria varied considerably and occurred during all parts of micturition. There was no dribbling, hesitancy or incontinence. A slight burning occasionally accompanied urination, and the patient occasionally suffered with nocturia and frequency.

Two months prior to admission, he was cystoscoped in the Out Patient Department and found to have a papillary growth of the bladder 1.5 cm. above the left ureteral orifice.

For three weeks before admission, the patient had an uncomfortable feeling in the left groin, and three days prior to entry he suffered with low midback pain lasting a day.

The past and family histories were irrelevant.

Physical examination revealed a well-preserved elderly man in no distress. Examination of the lungs and heart was negative except for a systolic murmur at the apex and over the aortic area. The abdomen was soft, and no masses were felt.

The blood pressure was 150 systolic, 92 diastolic. The temperature, pulse and respirations were normal.

The urine showed a +++ test for albumin, a few white blood cells and many red blood cells. There were no casts, and the concentration was good. A culture of the urine revealed a few colonies of *Staphylococcus albus*. Examination of the blood showed a red-cell count of 4,250,000 with 75 per cent hemoglobin, and a white-cell count of 6300 with a normal differential. The nonprotein nitrogen was 23 mg. per 100 cc., and the prothrombin was normal. The phenolsulfonephthalein test showed 35 per cent excretion in fifteen minutes, and an additional 15 per cent in half an hour. X-ray films of the skull and chest revealed no lesions resembling metastases.

Cystoscopy failed to reveal the papillary growth of the bladder previously seen in the Out Patient Department. An intravenous pyelogram showed delayed excretion of the dye, which faintly outlined the ureters and kidneys. The bladder outline was relatively smooth; there was a slight defect at the base consistent with an enlarged prostate. A retrograde pyelogram demonstrated bilateral pressure defects along the upper surfaces of both kidneys. However, the lower poles were relatively normal.

Operation was performed on the fourth day after admission.

Differential Diagnosis

Dr. George G. Smith: The question in this case is whether a twist of the torso sustained while the patient was walking, without any actual fall, could cause bleeding from trauma of a normal kidney. I do not believe it would. I think it might if the patient had renal stone or hydronephrosis or some pathologic condition in the kidney, which might be ripped or torn by any unusual motion.

The fact that the bleeding occurred during all parts of micturition is rather against a prostatic origin. Usually, blood from the prostate precedes urination — not always, but usually. Furthermore, there was no dribbling, hesitancy or incontinence, and nothing to substantiate a diagnosis of prostatic bleeding.

The answers to two questions would be helpful. Did the patient have any bladder residuum? Was the blood seen coming from the ureter or any particular source at the time of cystoscopy?

Dr. Tracy B. Mallory: Can you answer either of those two questions, Dr. Gens?

Dr. John P. Gens: There was no bladder residuum, and no blood could be seen coming from either ureter at the time of cystoscopy.

Dr. Smith: Was the patient bleeding at the time?

Dr. Gens: No.

Dr. Smith: It is a good thing to cystoscope patients with hematuria at the time there is blood in the urine, because then one can get an indication of the origin of the blood, which one often cannot get later on.

The fact that cystoscopy failed to reveal the papillary growth probably means that the alleged papilloma was a blood clot adherent to the bladder wall. It is very difficult to distinguish between an adherent blood clot and a papilloma that has been bleeding and is infiltrated with blood. With kidneys that show as good a renal function by nonprotein nitrogen and phenolsulfonephthalein tests as this man's kidneys did, one expects to see a very good picture of the kidney pelvis from the Diodrast or Neoskiodan, whichever one was used, unless he was not well prepared and had a great deal of fluid.

From the story to date, I have not the slightest idea what this man was operated on for. May we see the x-ray films?

Dr. Laurence L. Robbins: These are the films of the intravenous pyelograms, and as noted, there is no good visualization of the calyxes, pelves or ureters. The retrograde pyelogram, in contrast, shows these very obvious bilateral pressure defects in the outlines of both pelves. They are nearly symmetrical, but there is more involvement of the superior calyx on the left.

Dr. Smith: They are excellent and unusual films, I should say. The simultaneous bilateral occurrence of tumor is possible, but very unusual. It is much more likely that the patient had bilateral solitary cysts. The pelves and calyxes do not give the picture of congenital polycystic kid-

neys. Furthermore, the renal-function tests are too good for a case of polycystic kidneys in a man of this age. On the other hand, there is very definite pressure here, as Dr. Robbins has pointed out. The uppermost calyx is thinned out over a presumably smooth, symmetrical round tumor. It is quite possible to get marked hematuria from solitary cyst of the kidney, perhaps owing to interference with the blood supply, sometimes with rupture of the cyst. I should make a diagnosis of bilateral solitary cysts of the kidneys. I do not know which side I should operate on or why, but that is my diagnosis.

DR. MALLORY: Would you have operated on this patient on this evidence?

DR. SMITH: I suppose he could have had a tumor on one side and a cyst on the other, but it would be unusual to have the findings so symmetrical. If he had had successive episodes of bleeding and if observation had shown that he always bled from one side, I should be inclined to operate on that side. If it were a tumor, I should take out that kidney, and if it were, as seems more probable, a solitary cyst, I believe that removal of such a cyst would check the hemorrhage and that it would be to the patient's advantage to operate.

DR. WILLIAM B. BREED: Without that evidence, where would you operate?

DR. SMITH: As I say, I should want to know from which side the bleeding was coming. We do not know that from the history.

DR. BREED: Well, where are you going to operate — midline, right or left?

DR. SMITH: I should guess that the left kidney would be the one to pick because the upper calyx appears to be more involved in the x-ray film than the one on the other side, but I do not believe that is sufficient reason for selecting that side. I should insist on knowing which side was bleeding, and if this elderly man stopped bleeding and had no symptoms, I should leave him alone.

DR. J. DELLINGER BARNEY: What about bilateral adrenal tumors?

DR. SMITH: These lesions very evidently involved the structure of the kidney itself — the parenchyma of the kidney. So far as I know, adrenal tumors do not do that; they push the kidneys down but do not invade them.

DR. FLETCHER H. COLBY: It would add to the interest of the discussion if Dr. Smith were told the next step.

DR. MALLORY: We are prepared to do that. Dr. Gens, will you proceed?

DR. GENS: We never did know from which side the bleeding was coming. When the patient was admitted to the hospital, the gross hematuria stopped. The urine on both sides showed red blood cells. We decided to operate on the right side because we believed that the chance of cyst was greater on the left side since the calyxes were actually involved on the left side, whereas the tumor was pushing the pelvis or the calyx away on the right side, which would be more like neoplasm. Therefore, we operated on the right, came down on a cyst and treated it in the usual fashion.

DR. MALLORY: Would you then wish to do anything more, Dr. Smith?

DR. SMITH: I should assume that there was a cyst on the other side also, and unless he bled again or had further symptoms, I do not believe I should operate.

CLINICAL DIAGNOSIS

Cortical cysts of kidneys.

DR. SMITH'S DIAGNOSIS

Bilateral solitary cysts of kidneys.

ANATOMICAL DIAGNOSES

Solitary cyst of right kidney.
Papillary adenocarcinoma of left kidney.

PATHOLOGICAL DISCUSSION

DR. MALLORY: Suppose you go ahead with the story, Dr. Gens.

DR. GENS: About seven days later, although the patient had a perfectly uneventful immediate postoperative course, he once more had gross blood in the urine.

DR. SMITH: May I ask what you did to the right kidney?

DR. GENS: We excised the wall of the cyst. Then we decided to aspirate the left side but did not obtain anything but a few drops of blood, although we were fairly certain that we had touched the tumor mass in the left kidney.

DR. SMITH: But you knew the blood was coming from the left side?

DR. GENS: No.

DR. SMITH: I believe I should have looked in the bladder to see if the blood were coming from the left side.

DR. MALLORY: I think by exclusion it was believed that the hemorrhage must have been coming from the other side.

DR. SMITH: I think that is assuming too much; however, it probably was so. But I do not believe one can take anything that is easily demonstrated for granted. One could easily put in a small cystoscope and look at the ureters.

DR. MALLORY: Eventually, the patient was explored on the left side, an obvious tumor was found, and a nephrectomy performed. He made an uneventful convalescence. The tumor proved to be a papillary adenocarcinoma of renal origin.

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THE SPREAD OF INFANTILE PARALYSIS

It is difficult to correlate the fact that the incidence of infections of the upper respiratory tract is lowest and that of poliomyelitis — a disease in which the upper respiratory tract is generally considered to be the important, if not the only, portal of entry of the virus of infantile paralysis — highest during the late summer and early fall. This view concerning the origin of poliomyelitic infection has been based chiefly on the following facts: the virus of poliomyelitis has been demonstrated in nasal washings obtained in the early stages of the infection; the disease, during outbreaks, is considered to occur with more than the expected frequency in relation to tonsillectomy; and it is relatively easy to infect the susceptible monkey with the virus by intranasal inoculation.

Recent experimental work, however, indicates that the nasal passages may constitute a point of exit as well as of entrance for the virus of poliomyelitis. Thus, the virus has been recovered from the nasal secretions of monkeys infected by inoculation into the brain and even into the peritoneum. Furthermore, in monkeys inoculated intranasally, the virus disappears from the nose a few hours later and reappears there after a lapse of about forty-eight hours.

Increasing evidence is being brought forth to implicate the gastrointestinal tract as the route of infection in poliomyelitis. This, of course, is much more consistent with the seasonal incidence of the infection, and the long-known association of epidemics of the disease with the course of rivers and in relation to other bodies of water. Even as early as 1912, Kling and his colleagues,[1] in Sweden, reported the finding of the virus of infantile paralysis in the stools of human cases. This observation has been confirmed and extended sporadically by a number of workers, but intensive investigations based on this finding have been carried out only during the last three or four years.

Briefly, it has now become clear that the virus of poliomyelitis can be demonstrated quite readily in the stools of frank cases and of so-called "abortive cases." In fact, the virus can be found much more frequently and over much longer periods in the stools than in the nasal passages. The specific virus has also been identified in the stools of a large percentage of intimate, but healthy, contacts of cases, as well as in the sewage from homes and areas where the disease is prevalent. Furthermore, it has been shown to exist in the stools of patients and carriers for considerable periods. These findings have recently been reviewed by Armstrong[2] and by Paul.[3]

The ease of identifying the virus of poliomyelitis in stools has proved to be of the greatest help in epidemiologic studies of this disease. Re-

cently, Piszczek, Shaughnessy, Zichis and Levinson[4] reported the results of their studies of an outbreak in a suburban community in Cook County, Illinois. The outbreak involved 15 cases between June 4 and July 11, 1941. In 3 out of 4 patients, the virus of poliomyelitis was demonstrated in samples of feces collected about one month after the onset of symptoms. The virus was also found in the stools of at least 8 out of 48 contacts, but not in the feces of 25 healthy residents of the same community who had had no contact with the disease. In view of previous epidemiologic notions concerning the familial spread of the disease, it is noteworthy that the virus was found in the stools of 2 presumably healthy children who were in contact with adult members of their families who, in turn, became ill with poliomyelitis. The authors succeeded in isolating the virus from a supposedly healthy carrier two months after he had had contact with persons who became ill with poliomyelitis and one month after exposure to his father, who died of poliomyelitis.

The epidemiologic evidence brought to light by these studies seems to point strongly to a spread of the disease by apparently healthy carriers—a view that has been held for over twenty-five years by the Swedish workers, who supported the contention that the infection was spread by way of the gastrointestinal route.

All these findings are beginning to clarify the picture of the epidemiology of infantile paralysis. It is hoped that they will result in the development of effective measures for the control of this disease.

References

1. Kling, C., Wernstedt, W., and Petterssar, A. Recherches sur le mode de propagation de la paralysie infantile epidemique (maladie de Heine-Medin). *Ztschr. f. Immunitätsforsch. u. exper. Therap.* 12:316-323, 657-670, 1912.

2. Armstrong, C. The etiology of poliomyelitis. Lecture 2 in *Infantile Paralysis, 1941*, a symposium delivered at Vanderbilt University, April, 1941. 239 pp. New York: National Foundation for Infantile Paralysis, Incorporated, 1941. Pp. 37-55.

3. Paul, J. R. The epidemiology of poliomyelitis. Lecture 3 in *Infantile Paralysis, 1941*, a symposium delivered at Vanderbilt University, April, 1941. 239 pp. New York: National Foundation for Infantile Paralysis, Incorporated, 1941. Pp. 121-157.

4. Piszczek, E. A., Shaughnessy, H. J., Zichis, J., and Levinson, S. O. Acute anterior poliomyelitis. Study of an outbreak in west suburban Cook County, Ill.: preliminary report. *J. A. M. A.* 117:1962-1965, 1941.

DRUGS AND MENTAL DISEASE

It is encouraging that drugs and chemical agents are very minor causes of mental disease in Massachusetts: a recent study[1] indicates that of 115,000 patients cared for in Massachusetts mental hospitals between 1917 and 1937, only 841 cases seemed to have been associated with the use of drugs and chemicals. Of these, 36 cases were unverified, so that the actual number was 805 cases, or less than 0.7 per cent. These cases were diagnosed as "psychoses due to drugs and other exogenous toxins," except for cases labeled "without psychosis: drug addiction."

A comparison of these cases with those related to alcoholism originating in the same geographic area during the same period is interesting. According to Dayton,[2] alcoholism caused more than fifteen times as many entries to state mental hospitals as drugs. Furthermore, many of the patients using drugs also drank alcohol in excessive amounts and therefore might have been classified as alcoholic cases with equal validity: among first admissions related to drugs and chemical agents, 50.4 per cent of men and 25.5 per cent of women were intemperate users of alcohol, and Dayton found that 32.0 per cent of all men admitted with psychoses and 6.0 per cent of all women were intemperate.

It appears that users of drugs are not especially selective in their choice of pharmacologic comforts, since these patients took more than one drug (sometimes three or four) either concurrently or successively.

Many persons in the group stated that their use of drugs began when they were given drugs on prescription by a physician. They may have had the prescription refilled, or the physician may have failed to withdraw the drug when the specific need was over. A few began taking drugs on the advice of friends or relatives, but self-medication was a less important factor than therapeutic use under medical advice. Despite the limited number of cases represented, physicians should be aware of the possibilities that may follow the injudicious use of drugs.

Perhaps the most striking finding in the study, aside from the excessive use of alcohol, was the high incidence of abnormal personality traits and prepsychotic personalities. Many patients had histories of frank mental disease and previous admissions to mental hospitals for reasons unrelated to drugs. It might be assumed that their resort to drugs was a symptom of psychologic illness and that the intoxication or addiction was a result rather than the cause of illness.

The major drugs reported were as follows: opium and its derivatives, 363 cases; barbiturates, 208 cases; and bromides, 101 cases. Many of the cases were suicidal, accidental or due to industrial exposure and cannot be considered in relation to addiction — the standard classification groups cases of this type with all others related to chemical agents.

Among the facts emphasized in this study is the need for the physician to recognize the limitations of medication related to mental disease and to be especially aware that the uncontrolled use of certain drugs may precipitate mental disease in patients who are predisposed, who have histories of previous mental illness or who are inclined to have unstable personality trends. The need for additional investigation into the effect of drugs on personality is evident.

In addition, it is pointed out that the complications of drug addiction may be related to the faulty diets, the avitaminoses and the abnormal regimes of personal hygiene of drug users, as has been shown for alcoholic complications. The heterotropic quality of the addiction pattern in this group is of interest because it seems to demonstrate that one drug can be substituted for another and that, consequently, little additional evidence is needed to demonstrate conclusively that addiction is more basically associated with personality types than with specific chemical agents.

References

1. Moore, M., Raymond, A. F., and Gray, M. G. Drugs and mental disease: a review of 841 cases. *Confinia Neurol.* 4:238-271, 1941.
2. Dayton, N. A. *New Facts on Mental Disorders.* 486 pp. Springfield, Illinois: Charles C Thomas, 1940.

MEDICAL EPONYM

Rauchfuss's Triangle

Although Carl Rauchfuss (1835–1915) discussed "Die paravertebrale Dämpfung auf der gesunden Brustseite bei Pleuraergüssen (The Paravertebral Dullness on the Healthy Side in Pleural Effusions)" before the Pediatric Society of St. Petersburg in April, 1903, the first published account of his finding appears under the above title in *Verhandlungen der Einundzwanzigsten Versammlung der Gesellschaft für Kinderheilkunde in der Abteilung für Kinderheilkunde der 76. Versammlung der Gesellschaft deutscher Naturforscher und Ärzte in Breslau, 1904* (21:202–211, 1905). A portion of the translation follows:

> The clinical finding . . . is the triangular area of dullness, which can always be demonstrated by percussion or palpatory percussion, on the healthy side, along the vertebral column and extending upward to a height that depends on the extent of the pleural effusion, frequently as high as its upper level. The base of the triangle corresponds to the lower border of the lung, and its hypotenuse leaves the base at a distance of 2 to 5 cm. from the spinous processes (in childhood).

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON MATERNAL WELFARE

Case History: Post-partum shock followed by death

A twenty-eight-year-old primipara had had adequate prenatal care. The past history was irrelevant, and at no time during her pregnancy was elevation of the blood pressure or albuminuria noted. There had been no flowing, and the size of the pelvis was adequate. Labor started spontaneously and lasted twenty-six hours until full dilatation. Delivery was difficult because of a transverse arrest, but a living child was obtained. Immediately after delivery, the patient went into shock, the pulse rising to 160 (the blood pressure was not recorded). Extraction of the placenta offered no problem, and there was no unusual bleeding. Eight hours after delivery, the hospital authorities thought the patient to be in such poor shape that they took it on themselves to call an obstetric consultant, who stated that her condition was critical. Although there was no alarming bleeding at this time, she was in profound shock, presenting such typical signs as cold, clammy skin, rapid, weak pulse and low blood pressure. The consultant advised immediate transfusion, but this measure was not carried out at once because the patient's physician saw her soon afterward and

diagnosed the cause of her condition as dilated stomach. The stomach was washed out, and a Wangensteen suction apparatus set up. Ten hours after she had been seen by the consultant, the patient was transfused. This did little or no good, and death occurred shortly afterward.

Comment. This patient was taken care of by a general practitioner and not by an obstetrician. Transverse arrests in the hands of well-trained men are difficult but, of course, less difficult than in the hands of the unskilled. This condition of shock may well have been brought on by the ineptness of the operator. It is interesting that the hospital took it on itself, because it felt its responsibility to the patient, to summon an obstetric consultant. This is as it always should be, for after all, the hospital is in effect responsible for every patient it receives within its doors.

This death was ascribed to acute gastric dilatation; however, it is probable that shock was the true diagnosis. It is deplorable that transfusion was delayed ten hours after it had been advised. Now that plasma can be obtained commercially, it should be in the pharmacy of every hospital; if given promptly, it should greatly decrease the fatalities due to shock.

WAR ACTIVITIES

AMERICAN RED CROSS

Expansion of Blood-Procurement Project

Expansion of the blood-procurement project from an original 900,000 donors during the current fiscal year to a minimum of 2,500,000 has been requested by the Surgeons General of the Army and Navy, the American Red Cross has announced. This almost threefold increase will require approximately 50,000 volunteer donations a week. The eighteen blood-donor centers operated by Red Cross chapters in the more populous areas of the country are rapidly completing plans to increase their quotas, and a few additional centers will be opened to handle this augmented number of donations. In addition to dried plasma, blood collected is to be processed into human serum albumin.

Serum albumin as a blood substitute is a recent development for which Dr. Edwin J. Cohn, professor of physical chemistry, Harvard Medical School, is largely responsible. The process at present is regarded as a military secret, but it may be said that albumin is a protein extracted from blood, and some of the processing is conducted at temperatures below 32°F. As in the processing of powdered plasma, blood used in preparing serum albumin must be shipped in refrigerated containers and must reach the laboratory within twenty-four hours after being drawn. Unlike dried plasma, serum albumin, when prepared for shipment, is in solution. Nothing need be added before the transfusion is given. A unit of the serum consists of 25 gm. of albumin dissolved in 100 cc. of physiologic saline solution.

The albumin is put up in glass vials with rubber stoppers at both ends, in which tubes are inserted, and the transfusion is given intravenously. The vial and necessary tubes and needles are enclosed in a tin container, and complete directions for giving a transfusion are etched on each vial.

Serum albumin, according to present indications, will last for years without deterioration. Furthermore, it can be kept at temperatures ranging from slightly above freezing to approximately as high as 120°F. It requires about 3½ pints of blood to obtain one unit of albumin, but this disproportion is somewhat offset by the fact that, generally speaking, a unit is equivalent to two units of dried plasma as a blood substitute. The albumin will be used to supplement rather than replace dried plasma, since both products have certain distinctive advantages.

As of June 30 last, donations to the Red Cross blood-procurement project for the armed forces totaled 461,493, representing more than 80,000 above the 380,000 requested for delivery by that time by the Army and Navy.

In announcing the expanded program, the Red Cross explained that, until now, it has had to restrict quotas of the donor centers because of the limited capacities of processing laboratories. Several additional laboratories are expected to be participating in the program in the near future, however, and the capacity of those already processing blood is being expanded. The combined annual capacity of processors is expected soon to reach a total of at least 2,500,000 pints of blood, and may even go as high as 3,000,000, Red Cross officials stated. The eighteen donor centers participating in the national blood-procurement project are operated by Red Cross chapters in New York, Philadelphia, Baltimore, Rochester, Buffalo, Boston, Brooklyn, Cincinnati, Cleveland, Chicago, Detroit, Pittsburgh, Indianapolis, St. Louis, Milwaukee, Los Angeles, San Francisco and Washington, D. C.

MISCELLANY

WORK TOLERANCE FOLLOWING TUBERCULOSIS

Return of the arrested case of tuberculosis to safe and full economic efficiency is the final objective of treatment. This aspect of the care of the tuberculous patient, however, has received far too little thoughtful study. Fifty per cent of patients discharged from sanatoriums still die of tuberculosis within five years. This social waste must be stopped. Aitken (Aitken, A. M. The need for developing work tolerance following pulmonary tuberculosis. Read at annual meeting of National Tuberculosis Association, Philadelphia, May 6–9, 1942) calls attention to the need of a more scientific approach to the problem.

The original purpose of a sanatorium was largely the segregation of a patient with an infectious disease dangerous to his neighbors. Enough bacillary cases were cured or arrested through rest, fresh air and proper food to encourage the development of sanatoriums for the "early case" that held good hope for cure. Refined methods of diagnosis soon showed that the minimal case was a rarity and that prolonged bed rest was nearly always essential. This principle is still valid, even with the introduction of collapse therapy as an effective form of treatment. The criticism that healthy loafers were being made out of sick workers was too often justified.

Thereupon, occupational therapy crept in to relieve the tedium of enforced idleness, and then followed a more constructive approach, known by the awkward name of "rehabilitation," which included education and vocational training. Treating the disease while the patient is an invalid in the hospital is no longer considered sufficient.

Adequate care involves preparation for maximum social and economic adjustment when the disease is arrested or apparently cured.

This duty devolves on the sanatorium:

> As soon as an estimate of the disease processes is arrived at and the course of treatment decided upon, a beginning can be made in education. An early analysis of the patient's educational and occupational background, of his interests and aptitudes can be made and a course of training outlined. This can be made to synchronize with his medical treatment and other activities permitted, and it can be carried throughout the full length of stay of the patient in the sanatorium. As well, there are many of the facilities of the sanatorium which can be used for both training and physical rehabilitation. All the program requires is the co-ordination and co-operation of the various staffs of the hospital and occupational therapists who are willing to accept adult education as being a branch of occupational therapy.
>
> The appraisal of the ability of the individual to do some line of work begins with securing past-work history and continues throughout the period of training. Also the counseling of the patient and testing for special aptitudes by trained observers aids in appraising. It not only helps evaluation but it gives direction to effort, eliminating much time wasted by trial and error methods, and is most useful in creating interest and co-operation in patients.

Appraising the physical stamina of the patient to stand the strain of normal life is difficult. There are no clinical or mechanical tests as reliable measures of work tolerance. One cannot say just how many foot pounds of muscular energy a patient can safely expend, or how much mental strain he can endure without reactivating his disease. Furthermore, just how much energy a given job requires is but vaguely known. Job analyses are usually made on the basis of speed rather than foot pounds of energy required.

Present recourse, then, is the study of the patient as an individual during his stay in the sanatorium. Close observation will give an appraisal of his inherent resistance to breakdown from physical effort, nervous upsets or even intercurrent infection. With the knowledge thus gained, the trial method of graduated exercise should be undertaken with careful watching:

> Signs and symptoms of intoxication indicate overexertion and need for return to rest therapy. Rise in temperature, increase in pulse rate, fatigue and loss of weight, sputum changes in quantity and content, changes in sedimentation rate and blood count and later increase in pathology as shown by x-ray, suggest reactivation.
>
> In order to establish with more surety that a patient can withstand sustained efforts, a period of physical rehabilitation should be followed before discharge of the patient. Before it can be certain that the patient can lead a normal life and stand up to ordinary work conditions, sanatorium routine and cure hours should be broken. One of the hardest things for a patient is to discontinue the mid-day rest period. If he can be put on a full work schedule of forty hours a week for a few months before discharge and is able to play after work without undue fatigue, he should be able to do the same outside. This can be readily done in a sanatorium where there is a constant need for help and often to the advantage of the sanatorium.

In addition to the graduated exercise, test inferences may be drawn from x-ray studies of the characteristics of the disease during treatment, such as a tendency toward fibrosis, rapidity of healing and evidences of good resistance. On the other hand, very extensive disease, with reduced vital capacity, distortion of chest structures and possible cardiac embarrassment, obviously cause low work tolerance.

In connection with its rehabilitation program, for over ten years, Niagara Sanatorium (New York) has given close study to the problem of determining work tolerance. Although only about half the patients are considered to afford hope of effective vocational rehabilitation, careful study is made of every case, since whatever occupational therapy is possible is employed routinely. Patients have been given aptitude and personality tests by personnel from the National Tuberculosis Association, and the State Rehabilitation Department has made provision for the completion of courses in a number of cases.

Only modest claims are made for the results thus far achieved:

> It is true that the death rate in the sanatorium has remained unchanged, but the readmission rate has decreased, as have deaths of patients after discharge. This decrease in readmissions counterbalances the increased initial length of stay. Of fifteen patients who have been aided by state rehabilitation, only one has since broken down and this was the result of lobar pneumonia in a patient with a complete thoracoplasty. As well as the evident individual results we have obtained, the morale of the entire population has improved. Few patients leave now because of boredom. Also, it has given us an employment agency, not only for temporary help but for permanent employees who have been tried and their ability proved. Some of our most valuable employees are ex-patients, trained in the peculiarities of our set-up and most valuable in that they carry with them the patients' viewpoint and an understanding of patients' trials and tribulations.
>
> To summarize, a rehabilitation program can be developed in a small sanatorium with benefit to patients individually and collectively and with advantages to the sanatorium. Tolerance for selective work can be built up in patients, but the evaluation like that for determining disease status, being dependent upon personal judgment of the significance of the individual's reactions, is only approximate. The program can be carried on at no great cost to the community and over a period of time the community, as a whole, will be repaid many times over. — Reprinted from *Tuberculosis Abstracts*, August, 1942.

DEATH OF X-RAY PIONEER

Very soon after the discovery of the x-ray, a number of persons in Boston, who were interested in this new discovery, or plaything as it then appeared to be, gathered together what apparatus they could find in physical laboratories and began experimenting with this light. A few keen-minded physicians, sensing the importance of it, soon became very enthusiastic over the possibilities of its practical use in their profession. Among these was Dr. Francis H. Williams, then a member of the staff of the Boston City Hospital. During the following year, he made much use of this new method of diagnosis, the work being done in a small room in the basement of the hospital.

After a year's work in the poorly equipped laboratory, during which he had demonstrated to the profession the wide range of usefulness of the x-ray, Dr. Williams felt the necessity of the services of a professional photographer and mechanic and secured Ernest E. Fewkes, who had done much original work along these lines. The trustees of the Boston City Hospital later appointed Mr. Fewkes to the position of photographer and x-ray assistant to the hospital. That was June 18, 1893, only three years after the discovery of the x-ray. Mr. Fewkes's ability as a photographer, mechanic and all-round scientist enabled Dr. Williams to make great strides in his research work.

Mr. Fewkes received many x-ray burns while working with this light in the days before anything was known of the danger in being exposed to the rays. One by one, he lost three fingers on each hand as the result of burns sustained forty years ago. Later, internal burns developed, which finally resulted in death, which occurred at his home on Hyde Street, Newton Highlands, on July 16.

NOTES

The Massachusetts Society for Mental Hygiene announces the resignation of its medical director, Dr. Henry B. Elkind, effective August 12, when he entered active service as a major in the Medical Corps of the United States Army. Dr. Elkind had served as medical director of the society since 1925, and his able administration brought recognition of the society's leadership in the field of mental-hygiene education, not only in Massachusetts but throughout the Nation. During his directorship, which was characterized by resourcefulness, vision, and energy, he did much to extend the application of mental-hygiene principles to many important fields of community life, including child care, industry, social work, public-school education and teacher training. One particular branch in which he achieved special success is the application of statistics to the study of mental disease, and he is recognized as one of the foremost leaders in the country in the epidemiology of mental disease. Under Dr. Elkind's direction, the Massachusetts Society for Mental Hygiene has adapted its program to meet wartime needs, and he leaves it in a strong position to continue its contribution to the welfare of the community under the changed and difficult conditions of today.

Dr. Frank H. Washburn, of Holden, was recently elected governor of the American College of Chest Physicians at the annual meeting of the college. Dr. Richard H. Overholt, of Brookline, was elected second vice-president.

CORRESPONDENCE

A CORRECTION

To the Editor: It has come to my attention that you have reprinted my article "Unsocialized Medicine," in a special supplement to your journal. I was pleased to have my dissenting remarks included among the newspaper pieces lauding the new prepayment plan of the Massachusetts Medical Society, and was impressed by the profession's tolerance of my somewhat sharp criticisms.

However, you erred in identifying me as "president of the White Cross," and I'd appreciate your correcting this so as not to expose the White Cross (which supported the Massachusetts Medical Society legislation) to professional criticism for my personal views.

Francis H. Russell, now with the State Department in Washington, is president. Edward A. Taft, vice-president and Boston lawyer, has been acting president during his absence. I functioned only as president of the unofficial but enthusiastic Association of White Cross Members.

HAROLD PUTNAM

The Boston Globe
Boston

• • •

The *Journal* acknowledges its error of referring to Mr. Putnam as president of the White Cross. Incidentally, it is pleased to learn that the views of Mr. Putnam are purely those of an individual and not those of an organization — either the White Cross or the Association of White Cross Members. ED.

REPORT OF MEETING

BOSTON LYING-IN HOSPITAL JOURNAL CLUB

At a regular meeting of the Boston Lying-in Hospital Journal Club, held at the hospital on April 15, Dr. Edwin H. Place discussed "Contagious Disease in Relation to Obstetrics."

Although this is not a common problem, it becomes important in the individual case so far as care is concerned. There have been only 76 cases of communicable disease during pregnancy or immediately post partum in 50,000 admissions studied at the South Department of the Boston City Hospital. There were 8 deaths, but 4 of these occurred in cases of scarlet fever before the use of sulfonamide compounds, and this is still a serious problem. The task of prevention seems impractical; however, nearly all cases are preventable. It is no longer necessary for the obstetrician to refrain from treating streptococcal cases with the present knowledge of bacteriology and asepsis; but a physician assumes a grave responsibility if he treats these patients when such diseases can be avoided.

If the question arises of admitting for delivery a woman of a family in which scarlet fever has occurred very recently, the finding of a negative Dick test and negative cultures makes her safer than a routine admission. However, routine cultures are impractical because of the lack of knowledge of when or over how long a period prior to confinement they should be carried out. If cultures are contemplated, the staff should be periodically tested, for completeness. The finding of a few streptococcal or diphtheritic carriers in this manner is inconsequential when one considers the many that are missed. If such a program of safety is contemplated, it is better to immunize the carriers so that they will do no harm. On the whole, however, Dr. Place suggests that carriers be ignored, for they cannot be adequately treated and it becomes a burden to isolate them for weeks or months. He believes that pregnant women should be immunized, but most of the urban population is immune anyhow. In diphtheria, the prospective mother alone can be freed of suspicion by culture and Schick test.

If there has been mumps in the home, the woman in urban life will undoubtedly be immune, and if not, the opportunity for spread is remote. There are no good tests in this disease, and one can only rely on these probabilities. The giving of convalescent serum during the incubation period is sound. In chicken pox, the chances that an adult will contract the disease, even if exposed, are small. Measles is of little practical significance, since the children of urban mothers are usually immune.

In general, it has become the accepted policy of large

urban maternity hospitals not to admit any suspicious cases, and such women must be delivered at home under proper precautions or in a hospital for contagious diseases.

The incidence of communicable disease in the newborn is rare, especially those contracted in utero. Measles, smallpox and chicken pox are known to be contracted thus. Only one infant at the South Department of the Boston City Hospital has developed scarlet fever, and the children are always kept with their mothers. On the whole, this disease is rare under one year. Diphtheria is possible but at present unusual.

In discussing some slides, Dr. Place made several practical suggestions. Vaccination should never be carried out in the presence of eczema because of the danger of generalized vaccinia. Chicken pox is not contagious when the exanthem is pigmented. When this disease occurs during scarlet fever, all the lesions arrive at once, pass through the stages simultaneously and rapidly, and have the distribution of scarlet fever. The most valuable differentiation of smallpox and chicken pox is the rapidity of the changes in the former in contrast to even slight cases of the latter.

The discussion was opened by Dr. Stewart Clifford, who advocated specific admission rules for all outpatient departments. Dr. Arthur Hertig presented the case of a four and a half months' fetus with smallpox. He suggested that viruses may cross the placental barrier whereas bacteria do not, in general.

In conclusion, Dr. Place emphasized the rarity of intrauterine infections of this type. The question of whether coryza in an adult is actually a minimal whooping-cough infection may be settled by the proper use of cough plates and the finding of lymphocytosis. He believes that the benefit of convalescent serum is overemphasized. There is no center for this in Boston at present. So far as intrauterine immunity is concerned, it has been proved that the organisms of at least diphtheria, scarlet fever, whooping cough and mumps may pass across the placental barrier and, possibly, may even be concentrated thereby. But urban mothers are invariably immune to these organisms.

He made the following suggestions in answer to Dr. Clifford's request for specific outpatient-department rules. In diseases in which susceptibility tests are available, these may be carried out prior to confinement when time allows it. In cases of exposure to measles, chicken pox and mumps in the nonimmune mother, convalescent serum is not trustworthy enough to make admission more than a gamble. Such women probably should not be admitted if delivery is expected during the incubation period. If it becomes necessary to admit them, isolation is advised, and not much harm can ensue at worst.

BOOK REVIEWS

Surgery of the Heart. By E. S. J. King, M.D., M.S., D.Sc. (Melb.), F.R.C.S. (Eng.), F.R.A.C.S., Major A.A.M.C. 8°, cloth, 728 pp., with 4 plates and 268 illustrations. Baltimore: Williams and Wilkins Company, 1941. $13.50.

This first monograph of any size in the English language on surgery of the heart was written by a thoracic surgeon of Australia. It gives a wide survey of practically all surgical procedures that have been performed, or even suggested, on the heart and great vessels. In fact, the chief difficulty the reviewer finds with the book is that it is too expansive and includes a good deal of irrelevant matter concerned with medical rather than surgical aspects of heart disease. The volume could profitably be shortened by the omission of much of the material, which can be found in more detail in other books on cardiac physiology and disease.

The work is divided into two sections. The first takes up the anatomy, histology, physiology, embryology and pathology of the heart, with a discussion of roentgenology and electrocardiography. It is this part of the book particularly that might have been omitted or radically shortened to advantage. The second section deals in its introduction with the surgical approach to heart disease and with experimental investigations, and then considers, in order, the various diseases of the myocardium, endocardium, pericardium and great vessels. Of most practical value are the discussions of the treatment of cardiac wounds, foreign bodies and pericardial disease. There is a tendency to overstress surgical relief for myocardial and coronary heart disease, considering their relative unimportance or lack of success. It still remains to be proved how much good can be done for coronary heart disease by any surgical measures; too often, the common tendency for states of coronary insufficiency to correct themselves, no matter what therapy is carried out, is not recognized. There is an inadequate account of the important new operation introduced by Gross and Hubbard, of Boston, for closure of the patent ductus arteriosus.

The book is well arranged, and the frequent historical notes are of especial interest. An ample bibliography is appended to each of the section subdivisions, and should be very helpful for reference. There are numerous illustrations, a few of them in colors; many are helpful in relation to the subject at hand, but a considerable number might usefully have been omitted from a volume with this title. The book is attractively printed and bound.

Nostradamus: The man who saw through time. By Lee McCann. 8°, cloth, 421 pp. New York: Creative Age Press, Incorporated, 1941. $2.75.

In the last few months, there has been a revival of interest in Nostradamus, or at any rate in the prophecies of Nostradamus, which relate to certain world events up to 1999. There are those who pretend to discern, in the forecasts of this sixteenth-century astrologer, specific references to the humiliation of France, to Adolf Hitler and even to Pearl Harbor, although the attack had not occurred when this book when to press. Let those who believe take comfort; after these dark hours, the Axis powers are doomed to defeat. But it will not do to relax, merely on these prophetic grounds.

Two thirds of this book is devoted to a fictionalized biography of Nostradamus, and of the last portion to the predictions themselves, with elucidations of the particular modern events to which they refer. They need elucidation, for they are as obscure as the Apocalypse. But perhaps we have not the requisite faith. Miss McCann has; she is a hero-worshiper, at least of Nostradamus, whose life — he was a successful medical practitioner long before his ventures into world prophecy came to absorb all his energies — is painted as if he were one of the great. Some of us would be more inclined to rank him with Cagliostro or Elisha Perkins.

Symptoms in Diagnosis. By Jonathan C. Meakins, M.D., LL.D. 8°, cloth, 323 pp., with 54 illustrations. Boston: Little, Brown and Company, 1941. $4.00.

The importance of symptoms in diagnosis is well attested by this book, which is not a text of clinical medicine but rather a survey of symptoms as related to various syndromes and pathologic entities. It is written in a fascinating style, and its readability is enhanced by the elimination of all bibliographic references. Only a master of his field, as Dr. Meakins has proved himself to be, can assume this prerogative.

Dr. Meakins writes: "[The study of symptoms — as part of clinical medicine —] . . . needs young minds untrammeled by superstition and preconceived and fixed ideas which are the most deadly of all impediments to the advancement of medical knowledge. But with all this, there is a wonderful hope that through harnessing exact science in the way of sterol and physical chemistry and their sisters, electrophysics and biophysics, the day is not far hence when this tangle will be unsnarled and there will be gleams of light to illuminate the pathway to truth and understanding."

The pleasure of reading this work is akin to that which the bibliophile derives from browsing among books. Students and practitioners will find this volume particularly valuable.

Diseases of the Blood and Atlas of Hematology: With clinical and hematologic descriptions of the blood diseases, including a section on technic and terminology. By Roy R. Kracke, M.D. Second edition, thoroughly revised, reset and enlarged. 4°, cloth, 692 pp., with 54 color plates and 46 other illustrations. Philadelphia: J. B. Lippincott Company, 1941. $15.00.

That a second edition of Kracke's large and expensive book is forthcoming within a few years indicates its recognition as a standard text. The reviewer must confess, however, to a certain disappointment, which is perhaps based on his own idiosyncrasies regarding hematologic exposition. These derive from two fundamental and related concepts: that the hematologist is no longer a pathologist, but a clinician with a knowledge of the laboratory, and that hematology has advanced a long way from its previous preoccupation with the blood cells as the be-all and end-all of the subject. Hematology is now internal medicine, physiology and physiologic pathology, biochemistry and pathology. For these reasons, a work by a pathologist, however broad his outlook, must have its shortcomings, which indeed this volume possesses. Fully a third of the volume deals with variations in the morphology of the blood cells, with the result that the physiologic pathology and clinical features of some conditions are often not sufficiently stressed. There seems to be much absorption with systematization, terminology and classification, which savors of the ponderous Teutonic texts, formerly so popular. On the other hand, the pictures of blood smears and cells far outrank anything else in this country and for the *Atlas* alone, the book is worth owning. The lithography is unusually good.

There are excellent sections on agranulocytosis, in which the author did some of the pioneer work, and on leukemia. In the description of such preponderantly clinical conditions as pernicious anemia, idiopathic hypochromic anemia and the hemolytic anemias, however, the discussion is often pedantic, immersed in too many quotations from various authors and impeded by too much classification. For example, hemolytic anemia is discussed separately from hemolytic jaundice, although physiologically they are related conditions. The statement that the fragility of the red cells is always normal in acquired hemolytic anemia does not agree with numerous recent observations. Perhaps because the author is primarily a pathologist, considerations of therapy are often not only scant but misleading. This holds true not only for the treatment of acquired hemolytic jaundice, in which splenectomy is brushed aside as having little or no value, but also in the management of an acute case of idiopathic thrombopenic purpura. The statement is made that "splenectomy should not be attempted in the patient who has the disease in the acute form with severe hemorrhages, nor should it be done in those who are in a bleeding episode even though the course has been chronic." Unfortunately, many a patient has been lost — usually through cerebral hemorrhage — while physicians have been debating and temporizing. It is particularly in the acute form that splenectomy must frequently be done as an emergency measure. With a good surgeon, the risk is slight, and far less than that involved in temporizing.

The descriptions of thrombopenic purpura, polycythemia and Cooley's anemia are scant, and there is only a short paragraph on erythroblastosis foetalis. There is no discussion of the recently much discussed entity of leukoerythroblastic anemia associated with ectopic myeloid metaplasia. However, von Jaksch anemia, which has been regarded as defunct, rates a large paragraph. On the other hand, Custer's section on the bone marrow is good, as is the one on malaria and on hematologic technic.

In general, this is a good one-volume text on hematology, excellent for its histologic descriptions and pictures, but lacking somewhat in physiologic and clinical feeling. The printing and format are excellent, and the index and bibliography are good.

Eye Hazards in Industry: Extent, cause and means of prevention. By Louis Resnick. 8°, cloth, 321 pp., with 33 illustrations, 13 tables and 2 charts. New York: published for the National Society for The Prevention of Blindness by Columbia University Press, 1941. $3.50.

For the guidance of safety engineers, industrialists, workers and the medical profession in the prevention of eye injuries in industry comes this third volume in the past twenty-five years, published for the National Society for the Prevention of Blindness, and posthumously by the author Louis Resnick, who records his two decades of first-hand contacts with factories, mills and mines in the conservation of vision.

The first portion of the book deals with the problem of ocular hazards in industry, with especial reference to accidents, diseases of the eyes and defective vision. Each year in this country 300,000 workmen suffer eye injuries costing employers and employees the total of $200,000,000, 98 per cent of which are preventable. The author estimates that 80,000 workmen in this country have lost one eye and that 8000 workmen have lost both eyes as a result of preventable injury.

The second portion of the book deals with the solution of the problem through safety design, mechanical guards and reduced hazard at the machine; through safety devices to shield the eye; through adequate illumination; and through education of the workmen in the proper use of the appliances.

All statements are well supported by statistical evidence, and the book carries four appendices that contain useful information about industrial poisons hazardous to the eyes, minimum standards of illumination, and industrial welfare organizations.

(Notices on page ix)